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VOL. XX. No. 1

ARCHIVES OF OPHTHALMOLOGY.

ON THE CYLINDRICAL EQUIVALENT OF TILTED LENSES, THE PRISMATIC EQUIVALENT OF DECENTRED LENSES, AND THE EMPLOYMENT OF SUCH LENSES IN PRACTICE.

By WARD A. HOLDEN, A.M., M.D.,
ASSISTANT SURGEON N. Y. OPHTHALMIC AND AURAL INSTITUTE.

(*With five drawings.*)

I.—ON THE TILTING OF LENSES.

HISTORY.—Thomas Young, who discovered, measured, and corrected by a cylinder the astigmatism in his own eye, was the first to show that homocentric rays of light passing through a tilted spherical convex lens do not focus in a point, but in two lines perpendicular to each other, and, further, that within certain limits a tilted spherical lens before the eye has the effect of a spherical lens combined with a cylinder. Donders mentioned this principle in 1864 in his "Accommodation and Refraction of the Eye." Javal[1] first used it in practice in 1865. Javal's case was one of aphakia after cataract extraction, in which the cylinder $\frac{1}{16}$ brought the vision from $\frac{1}{4}$ to $\frac{3}{4}$, whereas by careful experiment the tilted spherical alone brought the vision to $\frac{1}{2}$ (Becker). Professor Pickering and Dr. Chas. H. Williams, while making physical experiments, noticed a disturbance when the lenses used were tilted, and, studying the matter thoroughly, found that a *tilted spherical has an increased spherical power as well as a cylindrical effect.* In their paper, "Foci of Obliquely Placed Lenses,"[2] are given two tables, one showing the increased refractive power in the horizontal meridian, the other showing the increased refractive power in the vertical meridian, of a lens tilted a series of angles on a vertical axis. These tables were made by careful experiment and verified by computation. Hay, in a paper "On the Increase of Refractive Power of a Plano-Cylindrical Lens when Rotated about its Axis,"[1] gave a formula deduced from one of the formulæ of Pickering and Williams, and explained the increase in refractive power by a diagram. Sous[2] did not accept Hay's explanation, and by a purely geometrical demonstration wished to show that there is not an increase in refractive power, but, on the contrary, a decrease. We shall look over these demonstrations in their order.

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[1] *Klin. Monatsbl.*, 1865, p. 339.

[2] Trans. American Acad. of Arts and Sciences, 1874–75, p. 300.

For the sake of clearness, with the tables of Pickering and Williams there will be presented also the methods by which they arrived at the formulæ for making the computations. In computing their tables a lens of 25.5 inches focal length was mounted on a rotating stand, and beyond it was placed a sliding screen having a horizontal and a vertical slit with cross hairs. Behind the screen was a gas flame, and a few inches in front of the lens a telescope. The lens was tilted on a vertical axis, the various angles given in columns 1, *i* indicating the angle of incidence of the rays, and the screen was moved first to the position in which the vertical slit was focused, and then to the position in which the horizontal slit was focused. Taking the focal distance of the lens F = 100, and the index of refraction of crown glass $n = 1.5$, column 2, Table I., gives the new focal distances for the vertical slit, and column 2, Table II., the new focal distances for the horizontal slit.

TABLE I.

i	F'	$\frac{df}{dn}$
0°	100	0.0
5°	98.9	0.0
10°	96.1	0.1
15°	91.2	0.2
20°	84.8	0.2
25°	77.	0.3
30°	68.4	0.4
35°	59.2	0.5
40°	49.8	0.6
45°	40.6	0.6
50°	32.	0.6
55°	24.1	0.6
60°	17.1	0.5

TABLE II.

i	F''	$\frac{df}{dn}$
0°	100	0.0
5°	99.9	0.0
10°	99.2	0.2
15°	97.7	0.3
20°	96.1	0.5
25°	93.7	0.6
30°	91.1	0.8
35°	88.3	1.0
40°	84.7	1.2
45°	81.1	1.5
50°	77.2	1.7
55°	73.2	1.9
60°	69.0	2.1

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[1] Trans. Amer. Ophth. Society, 1875, p. 319.

[2] "Traité d'Optique," 1881, p. 463.

Extracts from the original paper of Pickering and Williams follow, giving the working formulæ and the steps in their computation.

To represent these results theoretically, let us suppose the slits and lens so small, compared with their distance apart, that we may neglect all aberration except that due to the obliquity of the incidence. Considering first the case of the vertical slit, let Fig. 1 represent the section of a horizontal plane passing through the centre of the lens. Then let D represent the position of the slit when the emergent rays are parallel—that is, when A B is parallel to A′·C. Now $CD = F'$ is the new focal length to be determined. Call F the principal focal distance, *n* the index of refraction, *i* and *r* the angles of incidence and of refraction of the light on entering the lens, and *r*′ and *i*′ the corresponding angles on its emergence. Call also A the angle between the two surfaces of the lens at its edge, or of the two surfaces where pierced by the ray. Then, by the law of refraction, $\sin i = n \sin r$, and $\sin i' = n \sin r' = n \sin (r + A) = n \sin r + n A \cos r = \sin i + n A \cos r$, since $r' = A + r$ and sin A, being very small, may be regarded as equal to A. Again, $\sin i' - \sin i = \cos i\,(i' - i) = n A \cos r$, and hence $\frac{i' - i}{A} = \frac{-n \cos r'}{\cos i}$.

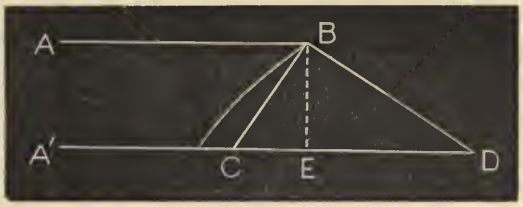

FIG. 1.

Now, in the triangle B C D we have $BDC = i - i' - A$, $BCD = 90° - i$, and B D sensibly equal to F′. Again $BC = FA(n - 1)$, for by the formula for lenses $\frac{1}{F} = (n - 1)\left(\frac{1}{R} + \frac{1}{R}\right)$, or $F = \frac{R}{2(n - 1)}$, but $A = \frac{2BC}{R}$ $\therefore$ $F = \frac{BC}{A(n - 1)}$, or $BC = FA(n - 1)$.

Since the sides are proportional to the sines of the opposite angles $BD : BC = \sin BCD : \sin BDC$, or $F' : FA(n - 1) = \sin(90° - i) : i - i' - A$, or $F' = F\frac{A(n - 1)\cos i}{i - i' - A}$. Dividing by

A, and substituting the value of $\frac{i - i'}{A}$ given above, we have

$$F' = F \frac{(n - 1) \cos^2 i}{n \cos r - \cos i} = F \frac{(n - 1) \cos^2 i}{\sqrt{n^2 - \sin^2 i} - \cos i} =$$

$$F \frac{(n - 1) \cos^2 i}{n^2 - 1} \left(\sqrt{n^2 - \sin^2 i} + \cos i\right) =$$

$$F \frac{\cos^2 i}{n + 1} \left(\sqrt{n^2 - \sin^2 i} + \cos i\right).^{1} \ldots\ldots\ldots\ldots\ldots\ldots (1)$$

By means of this formula the value of F′ was computed for every 5° for $n = 1.5$; and the results calling $F = 100$ are given in column 2, Table I. Column 3 gives the rate of change of F for small changes of n or $\frac{df}{dn}$. This is serviceable for computing the foci of lenses of various indices, but it is applicable only for values of *n* near 1.5 or 1.6.

The case of the horizontal slit is more complicated, since the rays no longer remain in one plane. Considering only those rays in the vertical plane passing through the axis around which the lens turns, and one point of the slit, we see that they will strike the lens at an angle of incidence about equal to i, will traverse it in a plane which we will call the plane of refraction, inclined to the first plane $i - r$, and finally emerge in a plane parallel to the first. The plane of refraction will intersect the lens along two circles, whose distance apart at the centre will be greater than the thickness of the lens in the ratio of cos r to 1 ; hence their radii R′ will be less than the radius of curvature R of the surfaces of the glass in the same ratio, or $R' = R \cos r$. Again, the apparent index of refraction n′ will be different, and since $\frac{1}{F} = \frac{2(n - 1)}{R}$ and $\frac{1}{F'} = \frac{2(n' - 1)}{R'}$ we have $F' = F \frac{n - 1}{n' - 1} \cdot \frac{R'}{R}$ $= F \cos r \frac{n - 1}{n' - 1}$. It therefore only remains to determine n′, the apparent index of refraction. As the problem is one in spherical trigonometry, suppose a sphere described around the centre of the lens and projected in Fig. 2, the eye lying in the axis of the lens prolonged. Let $CA = i$, the angle through which the lens has been turned, and $CE = r$, the corresponding angle of refraction. Then if the surface of the glass is vertical, as at the centre of the lens, the incident ray will be A C and the refracted ray C E. Next, suppose the surface slightly inclined by the amount $CD =$

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[1] In the original the formula has $n - 1$ as denominator of its fractional part. This is no doubt a typographical error. Both terms of the fraction in the preceding equation were divided by $n - 1$. The denominator in the last should therefore be $n + 1$.

B C = v as is the case for the upper and lower parts of the lens. A B = i′ will now be the angle of incidence ; and to construct the refracted ray, we have first the condition that sin i′ = n sin r′, and secondly, that the incident and refracted rays shall lie in the same plane with the normal B C D. To construct it, pass a plane through the normal B C and the incident ray A C, which will intersect the sphere along the great circles A B and F D ; on this, lay off D E′ = r′ such that n sin r′ = sin i′, but, as *v* is infinitesimal, it will be sensibly equal to i, and r′ to r. Now in the right-angled spherical triangle F C D, sin C D = sin D F sin C F D, or sin v = v = sin i × F, or $F = \frac{v}{\sin i}$; and in the triangle F E E′, sin E E′ = sin F E′ sin E F E′ or E E′= sin (1 — r) F, or substituting the value of F just found, $E\,E' = v\,\frac{\sin(1 - r)}{\sin i}$. Calling i″ and r″ the angles of incidence and refraction of the ray with regard to the section of the lens made by the plane of refraction ; then i″ will not equal B C but will be the angle, which when projected on the plane of the section of the lens, will be B C or i″ cos r — B C $\therefore i'' = \frac{v}{\cos r}$. Again E E′ is the angle through which the ray is bent or $i'' - r'' = v\,\frac{\sin(1 - r)}{\sin i}$ or subtracting

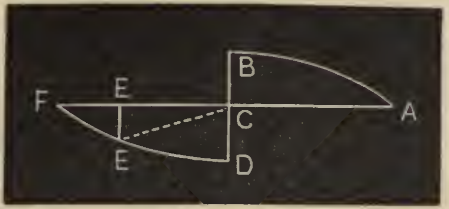

FIG. 2.

$$r'' = v\left[\frac{1}{\cos r} - \frac{\sin(1 - r)}{\sin i}\right] = v\,\frac{\sin i - \sin(i - r)\cos r}{\sin i \cos r}$$

but $n' = \frac{i''}{r''}$, which with the above values gives

$$n' = \frac{\sin i}{\sin i - \cos r \sin(1 - r)}$$

Substituting this value in the equation

$$F' = F \cos r\,\frac{(n - 1)}{(n' - 1)} \text{ gives}$$

$$F' = F\,(n' - 1)\,\frac{\sin i - \sin(i - r)\cos r}{\sin(i - r)}$$

This would be the focal distance if the rays on emerging remained in the plane of the section of the lens. But they pass into a plane inclined to this i — r, hence the observed focus F'' will be such that when projected on the plane of the section it will equal F', or $F'' \cos(i - r) = F'$. Hence finally

$$F'' = F \frac{\sin i - \sin(i - r)\cos r}{(n - 1)\ \sin(i - r)\cos(i - r)} \quad \ldots\ldots\ldots\ldots\ldots\ (2)$$

This last step may be open to criticism, but the close agreement with observation seems to justify it.

Hay gives as a formula, omitting the first term:

$$F \times \frac{\cos 2\varphi}{2(n \cos\varphi' - \cos\varphi)} \text{ in which}$$

F = focal distance of cylinder.
φ = its obliquity.
φ' = the angle of refraction corresponding to angle of incidence φ.
n = the index of refraction about equal to 1.53.

This is deduced from the formula of Pickering and Williams $F' = F \dfrac{(n - 1)\cos^2 i}{n \cos r - \cos i}$, the letters being changed, *n* being taken as 1.5 and as an equivalent to $n - 1 = .5$ in the numerator, 2 is placed in the denominator. This formula is correct.

Sous gives his demonstration as follows:

Let S be a luminous point situated on the principal axis S S′ of a spherical lens L′, and beyond its focus. That point will have its image at the point S′. The relation between the points S and S′, designating S O by p and S′ O by p′, will be

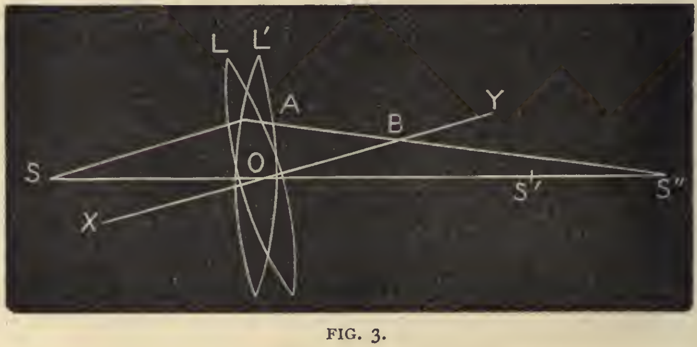

FIG. 3.

$$\frac{1}{p} + \frac{1}{p'} = \frac{1}{F} \text{ or } p' = \frac{p\,F}{p - F}.$$

If the lens be now tilted until it occupies the position L, the principal axis will be X Y and the point S will be situated on a secondary axis. To find the position of its image draw S A parallel to X Y. That ray passing through the focus B of the lens L will cut the secondary axis at S″ where the image of the point S will be found.

Let us determine the position of the point S″. In the triangle S″A S, O B being parallel to the base S A, we have

$$\frac{\text{O B or F}}{\text{A S}} = \frac{\text{S}'' \text{ O or p}''}{\text{S}'' \text{ O or p}'' + \text{O S or p}},$$

we have also $\dfrac{\text{A S}}{\text{O S or p}} = \cos \text{C O X}.$

Transposing into the first equation the value of A S found in the second and designating by α the angle S O X, angle of rotation of the lens, we get for the value of p″, $p'' = \dfrac{p F}{p \cos \alpha - F}$.

Let us compare the two values of p′ and p″. Cos α being a functional number less than unity and becoming smaller as the angle is greater, $p \cos \alpha - F$ is less than $p - F$; p″ is then greater than p′. The point S″ is then farther from the lens than the point S.

There is no positive error in this demonstration *per se.* Any demonstration of this sort employing the usual optical laws and formulæ will give an equally incorrect result. This may be shown in the following manner.

Let F and F′ (Fig. 4) be the principal foci of the lens L,

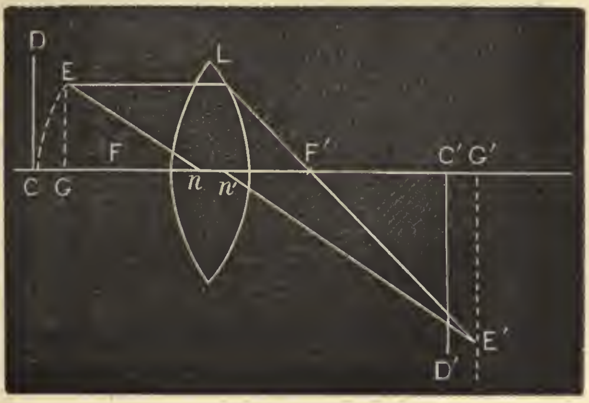

FIG. 4.

and let C and C′ be any pair of conjugate foci. Now consider the lens tilted, or, what is equivalent for our purpose, consider the point C moved from the axis, remaining, however, the same distance from the first nodal point of the lens. Let us suppose it moved to E and E n = C n. The problem now is to find the position of the image of E.

By the optical law the abscissæ of conjugate points outside the axis of a lens are also conjugate. That is, if C and C′ are conjugate foci, any point in the line D C will have its image in the line D′ C′. The abscissa of E is E G. The image of E must then lie at a point whose abscissa will intersect the axis at some point G′ which will be the conjugate focus of G. Now since C and C′ are conjugate foci the conjugate focus of G must lie farther from the lens than C′ at some point as G′. The image of E must lie then in the perpendicular erected on the axis at this point G′. To find the position of the image of E draw a line from E, parallel to the axis, to the second principal plane of the lens and from this through the focus F′, and where this line continued intersects the perpendicular erected at G′ will lie the point E′, the position of the image of E. The new focal distance is then n′ E′. In the figure we have the right-angled triangle n′ G′ E′; n′ E′ being the hypothenuse is greater than n′ G′ a side, and consequently greater than n′ C′ the primary conjugate focal distance. Hence the image of a point outside the axis of a lens lies farther from the lens than the image of an equidistant point on the axis. The refractive power of a lens is then decreased by the tilting.

These demonstrations, with results contrary to physical facts, show that the usual optical methods do not give even approximately correct results in all cases. Diagrams are given in the books for the purpose of showing more or less accurately the relations of various rays before and after refraction by a lens. The formulæ in the books, however, are computed only for rays which form small angles with the axis of the lens, and which emanate from a given point in that axis. These formulæ being applicable to such rays only, cannot be used at pleasure for rays having any relations whatsoever.

Methods of Finding the Effect of Tilting.—The increased refractive power of a tilted spherical convex lens may be determined experimentally with a fair degree of accuracy in the following manner: a disc with a slit receiving the handle of a test lens may be put into the clip of the trial frame and turned. The lens will then be rotated on a horizontal axis. Let the lens first be vertical and place a screen at its focus and a sharp image of a distant flame will be formed. Then tilt the lens the required number of degrees and a blurred image will be produced on the screen. Hold before the tilted convex lens that concave spherical lens and that concave cylinder, or the two concave cylinders, with axes horizontal and vertical, which will render the image sharp. These correcting lenses are the measure of the increased refractive power of the tilted lens. The lessened refractive power of a tilted concave lens may be determined in this manner, by holding before it in the tests a stronger convex spherical, so that an image will be formed on the screen. It will be found that the refractive power is lessened in the tilted concave lens in the same degree that it is increased in the tilted convex lens, consequently the same tables will serve for both. The positions of the new foci may be roughly determined as follows: the emmetropic eye of the observer on looking through the vertical lens will get a clear image of lines on the screen which is placed at the focus of the lens. Let the lens be tilted and all lines appear blurred. When the screen is brought nearer the lens, at some point the vertical lines will appear sharp. This distance from the lens to the screen will be the greater secondary focal distance. When the screen is moved still nearer, the horizontal lines will appear sharp. This distance from the lens to the screen will be the lesser secondary focal distance.

In the tables of Pickering and Williams, the two secondary focal distances have been computed for a single lens tilted at various angles. By means of their formulæ, however, the focal distances may be computed for any lens at any angle. For example, for finding the greater secondary focal distance of the lens $\frac{1}{40}$, tilted 30°, the formula is

$$F'' = F(n - 1)\frac{\sin i - \sin(i - r)\cos r}{\sin(i - r)\cos(i - r)} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (2)$$

The data are

$F = 40,$
$i = 30°,$
$r = 19° 28'$, from the formula, $n \sin r = \sin i$,
$n = 1.5.$

Then $F'' = 40\,(1.5-1)\,\dfrac{.5 - .1828 \times .9428}{.1828 \times .9831}$, and

$$F'' = 20\,\frac{.3276}{.1797} = 36.44$$

To find the lesser secondary focal distance of the same lens at the same angle, we employ the formula

$$F' = F\,\frac{\cos^2 i}{n+1}\left(\sqrt{r^2 - \sin^2 i} + \cos i\right) \ldots\ldots\ldots\ldots\ldots\ldots\ldots (1)$$

The data are as before. Then

$$F' = 40\,\frac{(.866)^2}{15+1}\left(\sqrt{(1.5)^2 - (.5)^2} + .866\right),$$

and $F' = 11.99\,(2.286) = 27.4$

After tables such as I. and II. have been computed by means of these formulæ for one lens at various angles, it is a simple matter to get the secondary focal distances for any lens. With a given angle the formula would vary for different lenses only in the value of F. The secondary focal distances of two lenses tilted at the same angle are to each other then as the primary focal distances. We can compute these secondary focal distances for any lens, using Tables I. and II., by a simple proportion. Table I. gives the lesser focal distance for varying angles, taking the primary focal distance F as 100. To find, for example, the lesser secondary focal distance of the lens $\frac{1}{40}$ at 30°, taking 100 from the 0° line and 68.4 from the 30° line of Table I., we have

$$100 : 68.4 : : 40 : F'', \quad 100\,F'' = 2736.0, \quad F'' = 27.36.$$

By the formula the value of F″ was found above to be 27.4. In the same manner the greater focal distances of any lens may be computed from Table II. Here follow computed tables of the secondary focal distances of a series of lenses at 10°, 20°, 30°, and 40° inclination.

TABLE III.

INCLINATION, 10°.

Focal length of lens, F	F″	F′
40	39.68	38.4
26	25.79	24.98
20	19.84	19.22
16	15.87	15.37
13	12.89	12.49
10	9.92	9.61
9	8.92	8.64
8	7.93	7.68
7	6.94	6.72
6½	6.44	6.24
6	5.95	5.76
5½	5.45	5.28
5	4.96	4.8
4½	4.46	4.32
4	3.96	3.84
3½	3.47	3.36
3	2.97	2.88
2½	2.48	2.40

TABLE IV.

INCLINATION, 20°.

Focal length of lens, F	F″	F′
40	38.4	35.92
26	24.98	21.04
20	19.22	16.96
16	15.37	13.56
13	12.49	11.02
10	9.61	8.48
9	8.64	7.63
8	7.68	6.78
7	6.72	5.93
6½	6.24	5.51
6	5.76	5.08
5½	5.28	4.66
5	4.80	4.24
4½	4.32	3.81
4	3.84	3.39
3½	3.36	2.96
3	2.88	2.54
2½	2.40	2.12

TABLE V.

INCLINATION, 30°.

Focal length of lens, F	F″	F′
40	36.44	27.36
26	23.68	17.78
20	18.22	13.68
16	14.57	10.94
13	11.84	8.89
10	9.11	6.84
9	8.19	6.15
8	7.28	5.47
7	6.37	4.78
6½	5.92	4.44
6	5.46	4.10
5½	5.01	3.86
5	4.55	3.42
4½	4.09	3.07
4	3.64	2.65
3½	3.18	2.39
3	2.73	2.05
2½	2.27	1.71

TABLE VI.

INCLINATION, 40°.

Focal length of lens, F	F″	F′
40	33.88	19.92
26	21.02	12.94
20	16.94	9.96
16	13.55	7.96
13	11.01	6.47
10	8.47	4.98
9	7.62	4.48
8	6.77	3.98
7	5.92	3.48
6½	5.50	3.23
6	5.08	2.98
5½	4.65	2.73
5	4.23	2.49
4½	3.81	2.24
4	3.38	1.99
3½	2.86	1.74
3	2.54	1.49
2½	2.11	1.24

The Applications of the Principle.

The practical points on which this subject bears are four, viz.: the wearing of tilted spherical glasses for the correction of ordinary compound astigmatism; the wearing of tilted sphericals in cases of aphakia with astigmatism; the change in the refractive power of the ophthalmoscope lenses when the instrument is tilted; and the change of refractive power in the convex lens behind the tilted otoscope mirror in examination of the membrana tympani.

The Tilting of Spectacle Lenses.—With regard to the first point, it is impracticable in ordinary compound astigmatism to tilt the glasses more than 20°, and then only on a horizontal axis. If, however, in compound hyperopic astigmatism, the cylinder is placed with its axis horizontal, which is not often the case, the correction may at times be made with a tilted spherical glass. If the refraction of the vertical and of the horizontal meridian corresponds to the reciprocals of two numbers in the same horizontal line in columns 2 and 3 of Tables III. or IV., or nearly corresponds to them, we can hope to make the correction by tilting a spherical lens of the focal length given in column 1 at the angle given above the table. For example, with the refraction of one meridian $\frac{1}{9.6}$ and of the other $\frac{1}{8.4}$ (line 6, Table IV.), or approximately accepting a spherical $\frac{1}{9.5}$ and a cylinder $\frac{1}{72}$, the correction can be made by tilting a $\frac{1}{10}$ lens 20°. In cases of compound myopic astigmatism, in which the axis of the cylinder is usually horizontal, the correction may often be made by tilting if the myopia is of considerable degree and the astigmatism proportionately slight.

The spherical lens tilted on a horizontal axis has the disadvantage that the refractive error is corrected only while the eye remains in a given horizontal plane. Suppose the upper edge of the glass tilted from the patient. Then if he look through the lower part of the glass, the visual axis of the eye comes to form a smaller angle with the axis of the lens, and the effect of the tilting is correspondingly lost. If he look through the upper part of the glass, the visual axis is at a greater angle with the axis of the lens, and the refractive power is increased. For this reason tilted spherical glasses are less adapted for distance than for reading.

In the astigmatism following cataract extraction the meridian of greatest refraction is in the direction of the line joining the puncture and counter-puncture made by the knife in the section of the cornea.[1] If the base of the corneal flap be horizontal, the axis of the correcting cylinder must be placed horizontal. If the base of the flap be inclined at an angle with the horizontal, the axis of the cylinder must be correspondingly inclined. Inasmuch as the axis of the cylinder is always horizontal or nearly so, the tilted spherical may be used to make the correction, provided that the astigmatism be not of high degree. Here follow tables showing the change in refraction of the lenses most used in aphakia, when tilted 10°, 15°, and 20°. The increase in spherical effect in the strongest lens at the greatest angle, *i. e.*, a lens of $2\frac{1}{4}$-inch focal distance tilted 20°, is only $\frac{1}{54}$. This increase is so slight in comparison with the strength of the lens (the spherical effect here would be $2\frac{1}{3}$ instead of $2\frac{1}{4}$) that it may be practically neglected. The tilted lens may then be considered as having only the spherical effect equal to the reciprocal of the number given in column 1. Column 3 gives the differences between the refractive powers of the horizontal and vertical meridians, *i. e.*, the cylindrical effect of the tilted lens. Column 4 gives the test-case glasses in inches corresponding nearest to the cylindrical effects given in column 3. In practice, then, column 4 gives the cylindrical effect produced by tilting the corresponding spherical in column 1, at the angle given above the table.

FOR 10° OF TILTING.

1.	2.	3.	4.
Sphericals in inches.	New focal distances.	Cylindrical effect.	Nearest corresponding test-glass.
$4\frac{1}{2}$	4.46–4.32	$\frac{1}{140}$	160
4	3.96–3.84	$\frac{1}{126}$	80
$3\frac{1}{2}$	3.47–3.36	$\frac{1}{106}$	80

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[1] Haase, *Wiesbaden klin. Beobachtungen*, iii., p. 116.

1. Sphericals in inches.	2. New focal distances.	3. Cylindrical effect.	4. Nearest corresponding test-glass.
3¼	3.22–3.12	$\frac{1}{100}$	80
3	2.97–2.88	$\frac{1}{95}$	80
2¾	2.72–2.64	$\frac{1}{89}$	80
2½	2.48–2.40	$\frac{1}{74}$	80
2¼	2.23–2.16	$\frac{1}{88}$	80

FOR 15° OF TILTING.

1. Sphericals in inches.	2. New focal distances.	3. Cylindrical effect.	4. Nearest corresponding test-glass.
4½	4.39–4.10	$\frac{1}{62}$	52
4	3.90–3.64	$\frac{1}{54}$	52
3½	3.41–3.19	$\frac{1}{49}$	52
3¼	3.17–2.96	$\frac{1}{45}$	40
3	2.93–2.73	$\frac{1}{29}$	40
2¾	2.68–2.50	$\frac{1}{27}$	40
2½	2.44–2.28	$\frac{1}{34}$	32
2¼	2.19–2.05	$\frac{1}{32}$	32

FOR 20° OF TILTING.

1. Sphericals in inches.	2. New focal distances.	3. Cylindrical effect.	4. Nearest corresponding test-glass.
4½	4.32–3.81	$\frac{1}{40}$	40
4	3.84–3.39	$\frac{1}{28}$	26
3½	3.36–2.96	$\frac{1}{24}$	26
3¼	3.12–2.75	$\frac{1}{23}$	22
3	2.88–2.54	$\frac{1}{21}$	22
2¾	2.64–2.33	$\frac{1}{19}$	20
2½	2.40–2.12	$\frac{1}{18}$	18
2¼	2.16–1.90	$\frac{1}{15}$	16

For a simple working table we may combine these three:

Sphericals in inches.	Test-case glass in inches corresponding to cylindrical effect of tilting.		
	10°.	15°.	20°.
4½	160+	52—	40
4	180+	52+	26—
3½	80—	52+	26+
3¼	80—	40—	22—
3	80—	40+	22+
2¾	80—	40+	20+
2½	80+	32—	18
2¼	80+	32	16+

The aphakic eye is probably practically the only one with which tilted sphericals would be much used in practice. As to the range of application, the paper of Scimemi[1] gives us the necessary data. He examined with the ophthalmometer of Javal and Schiötz, 146 eyes from which cataracts had been extracted. He found the horizontal diameter of the cornea lessened in length, and the vertical diameter increased in length after the extraction, the refraction being then less in the vertical meridian than in the horizontal. This astigmatism is then contrary to the usual corneal astigmatism, and may correct it.

The amount of astigmatism is markedly increased by accidents in and following the operation, such as prolapse of vitreous, iris, or lens capsule, enlargement of the section by the scissors, opening of the wound on a later day, or suppuration in it; the amount varies, also, according to the section and the manner of extraction used. In general he found the astigmatism, one month after the extraction, rarely as great as 3 *D*. The amount then present decreased until the second or third month, at which time it becomes permanent.

The Tilting of the Ophthalmoscope.—It might be supposed that the usual increase in refraction in tilted sphericals, would be influenced in the case of the ophthalmoscope, by the fact that the rays must pass through two small apertures —the perforation in the instrument, and the pupil of the observer. But the increase in refraction is the same here as elsewhere. It is easy to convince one's self by trial with the tilted ophthalmoscope, that the distances at which horizontal and vertical lines are clearly seen, are those given in the tables, for that lens and angle of inclination. This concerns the question of the tilting mirror. Loring, one of the chief advocates of the tilting mirror, says:[2] "In regard to the advantage of a mirror set at an angle for the use of the upright image, there can be no possible doubt in the mind of any one who has once seen the difference, in the ease of illumination and the clearness and brilliancy of the picture. By its use, a large quantity of light is saved, since the correcting glass, instead of being at a considerable angle with the axis of vision, and, therefore, in a position favorable to a largely increased loss of light through reflection from the surface of the glass, is nearly at right angles to it, by which all the light possible is saved." This explanation of the reason for the loss of light is manifestly incorrect. Jaques,[1] in a paper on "Light Transmitted by One or More Plates of Glass," has shown that if a given illumination be taken as 100, the amount of light transmitted through one glass plate is 89.5 when the glass is perpendicular to the axial ray, 89.5 when the glass is tilted 10°, 88.5 when tilted 30°, and 86.3 when tilted 45°, the amount of light lost by tilting being infinitesimal. With two glasses in the primary position, the transmitted light is 81, and the amount lost by tilting is very small. The brightness of the image, with the ophthalmoscope tilted, is diminished, not because the lenses are inclined, it being diminished almost as much when there is no lens before the perforation; but because a smaller number of rays reach the eye through the oblique perforation, whose size as regards the plane perpendicular to the visual axis, has become smaller. This, however, is a matter of little importance. If the position of the light is correct, the ophthalmoscope need never be tilted an appreciable amount. The principal cause of the greater brilliancy and clearness of the picture that Loring found, is, that when both instruments are held very near the examined eye, the tilted mirror receives light on a greater portion of its surface than the fixed mirror, and more light is thrown into the eye. The tilting mirror has the advantage that the instrument having it may be held nearer the examined eye than the instrument with a fixed mirror, and still furnish a sufficient illumination. If, however, the light be placed properly, that is, so that the rays fall on the external margin of the orbit, and are not cut off from it by the cheek—and if the observer's eye be so placed as to bring it as near as is practicable into line with the light and the examined eye, an ophthalmoscope with a fixed mirror will furnish a good illumination, and will not be tilted an appreciable amount. It may be necessary to hold it a little farther from the eye than an instrument with a tilting mirror, but this distance may be allowed for in estimating the refraction, and the observer may be sure that the lenses are looked through squarely, and that they have only the refractive power that is marked on the instrument. With a tilting mirror, little attention is given to the position of the light; and the instrument being held as near the eye as possible, is often unconsciously so tilted that a marked cylindrical effect is produced; and vessels running in one direction appear sharp, while those at right angles appear blurred, in cases of simple hyperopia or myopia. In refraction tests, the position of the light is more important than the position of the mirror. With a properly placed light, *i.e.*, $1\frac{1}{2}' - 2'$ behind the patient, as accurate a result may be obtained with the fixed mirror as with the tilted mirror. The tilting mirror complicates the instrument and is inconvenient, while it would not seem to have the decided advantages that it is supposed to possess.

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[1] Scimemi: "Sull' astigmatico corneale in seguito ad estrazione di cateratta." *Annali di Ottalmologia*, p. 299, 1889.

[2] "Ophthalmoscopy," p. 249.

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[1] Proceed. American Acad. of Arts and Sciences, 1874–75, p. 339.

With cylindrical lenses in the ophthalmoscope, when the axis of the cylinder is horizontal, no increase in refraction will be caused by tilting the instrument. When the axis is vertical, tilting will cause an increase in the refractive power, and the new focal distance of the cylinder may be found in column F′ of the table for that angle, opposite the number in column F which is equal to the focal length of the cylinder.

The Tilting of the Otoscope Mirror.—For the examination of the membrana tympani many aurists place a $+\frac{1}{10}$ spherical lens behind the perforation in the otoscope mirror. Since the light comes from behind the patient's head, the reflected rays from the mirror form an angle with the incident rays of from 20° to 40°. The mirror must then be tilted at an angle of from 10° to 20°. If it be tilted 20° the increased refractive effect of the lens is such that the mirror must be brought from 10 inches, its usual distance from the membrana tympani, to 9.6 inches before vertical lines can be seen sharply, and to 8.5 inches before horizontal lines can be seen sharply, and at no distance will the eye have a clear image of the membrane. The cylindrical effect of a $+\frac{1}{10}$ spherical tilted 20° is $\frac{1}{8.48}-\frac{1}{9.61}=\frac{1}{72}$. The cylindrical effect when tilted 10° is $\frac{1}{9.61}-\frac{1}{9.92}=\frac{1}{309}$. A $\frac{1}{160}$ cylinder with the $\frac{1}{10}$ spherical will not lessen the sharpness of an image, a $\frac{1}{80}$ cylinder will lessen it materially. Placing then a $\frac{1}{160}$ concave cylinder, with axis vertical, in combination with the $+\frac{1}{10}$ spherical, the cylindrical effect will be less than $\frac{1}{160}$ at every angle of inclination between 0° and 20°, and a clear image will be obtained for all distances under 9.6 inches.

With a simple spherical lens behind the mirror, the position of the light and the inclination of the auditory canal are of importance, and the smaller the angle between incident and reflected rays, the less is the mirror tilted and the sharper will be the detail of the image seen.

Recognizing the considerable cylindrical effect of tilted spherical lenses, it is evident that in order to obtain a maximum of vision in patients wearing spherical lenses, care must be taken to have the lenses in a plane perpendicular to the visual axis, which plane for distant vision is not the same as that for reading.

II.—ON THE DECENTRING OF LENSES.

We may complete the study of the subject of spectacle lenses with axes not coinciding with the visual axes, by considering the decentring of lenses for prismatic effect, in which case the lens undergoes a lateral or vertical displacement, while its axis remains parallel to the visual axis. Late text-books have given this subject a closer attention than the bare mention of the fact that decentring a lens produces deviation of the rays passing through it. Maddox[1] gives a table adapted from an earlier thesis, showing the prismatic effect of decentring any lens, the table being computed by means of the formula $\tan x=\frac{d}{f}$, which we shall consider later. Berry[1] gives a demonstration of the deviation, and also a formula for computing it, d = f tan Δ, which is identical with that of Maddox.[2]

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[1] "The Clinical Use of Prisms; and the Decentring of Lenses," London, 1889, p. 49.

It is possible, however, to demonstrate this deviation very simply, and to make formulæ so condensed and so easy of application that they may be readily employed in practice.

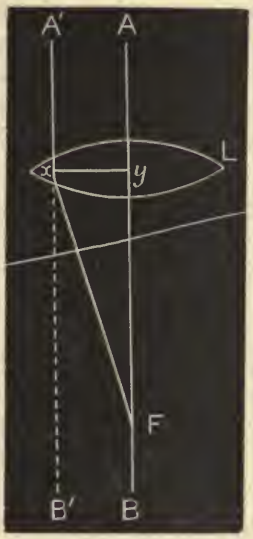

FIG. 5.

Let L (Fig. 5) be a lens with axis A B and its principal focus at F. A ray parallel to the axis, meeting the lens at any point, will be deviated and pass through the point F. Let A′ x be such a ray meeting the lens at x, undergoing there deviation and passing through F. The angle B′ x F is then the angle of deviation produced for a ray parallel to the axis, meeting the lens at the distance x y from its centre. For practical purposes, this angle B′ x F may be considered the angle of minimum deviation at the point x. Since A′ B′ and A B are parallel, angle x F y is equal to angle B′ x F.

We have then a right-angled triangle x F y in which y F is the focal distance of the lens, x y the distance which the lens is decentred, and angle x F y the angle of deviation of the ray. For x y putting d, and for y F putting f, and for angle x F y putting angle x, we have $\tan x = \frac{d}{f}$ and $d = f \tan x$. For a given lens and angle of deviation the decentring necessary may thus be computed. Angle x is, however, the angle of deviation.

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[1] "Diseases of the Eye," Am. edition, Philadelphia, 1889, p. 493.

[2] See also a paper by Schiötz and one by Prentice, these ARCHIVES, Nos. 2, 3, 1890.

In a prism of crown glass having a refractive index 1.5, the angle of minimum deviation or dioptric angle is half the physical or refracting angle of the prism. Prisms are still marked for the most part with the number of degrees of their physical angle, notwithstanding the efforts to introduce Jackson's better plan of numbering prisms according to their dioptric angle or deviating power.[1] Maddox, throughout his table, gives the deviating angle of the prisms. The formula for finding the decentring necessary to produce the effect of a given prism, as prisms are now numbered, is $\tan \frac{1}{2} x = \frac{d}{f}$.

Prisms stronger than 4° before each eye cannot conveniently be used in spectacles. We need consider then for the various lenses only that decentring which will give a deviation equal to that produced by prisms of 1°, 2°, 3°, and 4°. If the formula be worked out for a lens of 1-inch focal distance, having a deviation equal to that of a 1° prism, the amount of decentring found may be taken as a unit, and for any other lens, the descentring necessary for producing the effect of a 1° prism, may be found by multiplying this unit by the focal distance of the lens in inches. Such units may also be found for prisms 2°, 3°, and 4°.

For the effect of a 1° prism, $x = 1°$, $f = 1$ inch $= 25$ *mm*, $d = f \cdot \tan \frac{1}{2} x$, $d = 25 \times .00872 = .218$ *mm*.

For the effect of a 2° prism, $x = 2°$, $f = 25$ *mm*, $d = 25 \times .0174 = .435$ *mm*.

For the effect of a 3° prism, $x = 3°$, $f = 25$ *mm*, $d = 25 \times .02618 = .654$ *mm*.

For the effect of a 4° prism, $x = 4°$, $f = 25$ *mm*, $d = 25 \times .0349 = .872$ *mm*.

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[1] The numbering of prisms according to their metre-angles or dioptry-angles, lately advocated by so many writers, seems to me a cumbrous, inconvenient, and inaccurate system ; and I have yet to see a valid argument in favor of its adoption.

A table may be made thus:

In a Lens of 1 *Inch Focal Distance.*

Effect of prism.	Obtained by decentring.
1°	0.22 *mm.*
2°	0.43 *mm.*
3°	0.65 *mm.*
4°	0.87 *mm.*

To find the decentring necessary to produce the effect of a given prism in any lens, *multiply* the unit for that prism by the focal distance of the lens in inches. Example: The effect of a 2° prism wished in the lens $\frac{1}{15}$. Decentring necessary 0.43 *mm* × 15 = 6.5 *mm.* Or since the amount of decentring for producing the effect of any prism is equal to .22 multiplied by the number of that prism, we may take simply—*lens* 1 *inch,* 1° *prism by decentring* 0.22 *mm. For the effect of any prism, multiply* 0.22 *mm by the number of that prism, and for any lens multiply this product by the focal distance of the lens.*

Such a table may be made also for lenses numbered after the metric system by taking the decentring required in a 1 D lens as the unit. These units may be obtained by multiplying each of the units in the preceding table by 40, the focal distance in inches of a lens of 1 D.

In a Lens of 1 *Dioptry.*

Effect of prism.	Obtained by decentring.
1°	8.7 *mm.*
2°	17.4 *mm.*
3°	26.2 *mm.*
4°	34.8 *mm.*

To find decentring necessary to produce effect of a given prism in any lens, *divide* the unit for that prism by the number of dioptries of the lens. Example: The effect of a 3° prism wished in a lens of 7 D. Decentring necessary, 26.2 *mm* ÷ 7 = 3.7 *mm.* Or take—*lens* 1 D, 1° *prism by decentring* 8.7 *mm. For the effect of any prism multiply* 8.7 *mm by the number of that prism, and for any lens divide this product by the number of dioptries of the lens.*

The decentring for cylinders and combinations containing cylinders may be computed by using the refractive power of the horizontal meridian of the lens. To produce the effect of prisms base in, convex lenses should be displaced inward, and concave lenses outward.

Applications of the Principle.

The actual geometric decentring of a lens for prismatic effect is a matter more complicated and expensive than the grinding of a combination of lens and prism. The application which may be made of the tables given above is in the displacement of the usual lenses by spreading the spectacle rims farther apart or by bringing them closer together than they stand when centred, which is a much simpler procedure than combining a prism with the lens. This displacement may conveniently go as high as 7 *mm* for each eye. The range of application will then be seen from the following table showing the prismatic equivalents of various lenses displaced 6 *mm*.

Lens.	Displaced.	Has effect of prism.
1 *D*	6 *mm*	.75°
2 *D*	" "	1.5°
3 *D*	" "	2.°
4 *D*	" "	2.75°
5 *D*	" "	3.5°

Displacing each lens 6 *mm* produces the effect of the prism in the right-hand column before each eye. In this manner the lack of muscle balance may be totally or partially corrected, according to the lens used and the prismatic effect desired. Since the amount of displacement can be easily changed at any time, this is a convenient method of trying prisms temporarily.

What are the objections to this procedure, and are they valid? Swanzy[1] objects to the decentring as being less accurate than combining a prism with the lens. Without a table or formula for finding the prismatic equivalent of decentring, it is a loose method and one not to be commended, but, having these, it must needs be as accurate as the other.

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[1] "Diseases of the Eye," p. 422.

Mauthner[1] objects that the prismatic effects obtainable are too small to be of use, but gives no figures to support his statement.

Berry, speaking of displacing two + 5 D lenses each 6 *mm* inward to ease convergence to the extent of 1 metre angle, the interocular distance being 60 *mm*, continues :[2] "A rather greater effect would be got by combining prisms of 2° with the spherical lenses, so that decentring has evidently only a limited practical application." But by his formula, when $d = 6$ *mm* and $f = 3$ inches or 200 *mm*, $\tan \Delta = \frac{6}{200}$, and $\Delta = 1^\circ\ 44'$, or nearly 2° of deviation for each glass, and the prisms which would produce this deviation are prisms of nearly 4°, the strongest prisms ordinarily prescribed. He has here apparently confused the deviating angle and the refracting angle, and 6 *mm*, or ¼ inch, is about the greatest displacement that would ever be required with a 5 D lens, and the practical application of decentring is not so limited by half as he presumes it to be.

The Centring of Lenses.—If the subject of the decentring of lenses is worth our attention, the subject of the proper centring of lenses is equally so. The objection to a decentred glass is that it has the effect of a prism before the eye, and alters the condition of the muscle balance.

Let us first consider the effect of prisms before eyes with a normal muscle-balance. A prism of 1° in many cases, a prism of 2° in every case, placed base in or out before one eye, will cause a perceptible deviation of the eyes on alternate covering. The muscle balance is disturbed. With a patient having muscular insufficiency, if the prism which corrects the insufficiency be held before one eye, the eyes will show no deviation on alternate covering.

Pince-nez glasses are almost never centred, bowed spectacles seldom, if the measurements be left to the optician. A hyperopic patient, for example, shows at the consultation a normal muscle-balance with the correcting lenses. He gets the glasses from the optician, and when he returns the eyes show a deviation on alternate covering, when he fixes a point in the distance. The glasses then are not centred and the muscle-balance is affected. In many cases this may cause no asthenopia, but there is reason to believe that many of the cases of asthenopia persisting after glasses have been prescribed, are due to the prismatic effect of decentred lenses, which have relieved perhaps an accommodative asthenopia to cause a muscular one.

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[1] "Optische Fehler," p. 849.

[2] *L.c.*, p. 494.

It is not a simple matter to say whether glasses are accurately centred to the patient's pupils. The alternate covering, with distant fixation for distance glasses and fixation at the reading distance for reading glasses, is a ready method of finding, not whether the glasses are centred for the pupils, which is of cosmetic interest only, but whether they are so placed that the muscle balance is preserved. If the muscle balance were normal, the eyes would show no deviation, on alternate covering, only when the glasses were centred for the visual axes. If there existed an insufficiency of the interni or externi, the eyes would show no deviation only when the glasses were so placed that their prismatic equivalents corrected the insufficiency.

What use may we make of this test in practice? If the eyes show no deviation on alternate covering, the glasses may be left as they are, unless they are so placed as to narrow inconveniently the binocular field of vision. If the eyes show a deviation on alternate covering, find that prism which will restore the balance and prevent the deviation. From the above formulæ compute the displacement necessary to produce the effect of the prism found. Dividing the effect between the two eyes, let the lenses be displaced outward or inward, not to exceed 7 *mm* each from the pupillary centres, until the whole prismatic effect is produced, or that part of it which one may be able to produce in the given case without excessive displacement.

If, for example, the prism found were one of 3° base in, and the lenses were + 4 D, as shown above, the displacement required would be 8.7 *mm* × 3 ÷ 4 = 6.5 *mm*, and the optical centres of the two glasses should be brought 6.5 *mm* nearer each other, *i. e.*, each lens should be displaced 3.2 *mm* inward. The test for distance glasses should be made with distant fixation, for reading glasses with fixation at the reading distance, it being remembered that glasses centred to the pupils for the reading distance are from 2–3 *mm* nearer together than those centred for distant vision.

The purpose of this paper, the subject of which was kindly suggested to me by Professor Knapp, was to demonstrate mathematically these changes in the function of lenses whose axes do not coincide with the visual axes; further, to show in what circumstances these changes are to be avoided, and in what circumstances they may be utilized, and to give such formulæ and tables as may make it possible to employ them in our routine practice. Since, within the limits of their application, the tilting and the decentring of lenses are procedures simpler than those now commonly employed, it is to be hoped that these may come to have a more extended use.

A CASE OF NEURO-PARALYTIC KERATITIS, WITH MICROSCOPICAL EXAMINATION OF THE DISEASED EYE.

By G. E. De SCHWEINITZ, M.D.,

OPHTHALMIC SURGEON TO THE PHILADELPHIA AND CHILDREN'S HOSPITALS; OPHTHALMOLOGIST TO THE INFIRMARY FOR NERVOUS DISEASES.

SINCE the days when Herbert Mayo showed that section of the trigeminus within the cranium produced insensibility of the eye, and Magendie, in 1824, demonstrated that division of this nerve in rabbits resulted in anæsthesia of the globe, with inflammation and sloughing of the cornea, physiologists, ophthalmologists, and neurologists have undertaken a large amount of experimental research in the endeavor to definitely prove the cause of the corneal lesion. Even at the present day experimenters and clinicians are not in accord, and an examination of the statements in regard to the etiology of neuro-paralytic keratitis found in the standard text-books leaves the reader in doubt whether to accept that theory which ascribes the disease to a trophic change, or that which attributes it to the lessened power of resistance which the cornea, in its insensitive condition, presents to external injuries, or to believe, with Gowers ("Diseases of the Brain," p. 90), that "this neuro-paralytic ophthalmia, as it has been termed, probably depends on the irritation of the nerve by the lesion rather than on the anæsthesia, or on the mere loss of nerve influence."

The case which I am about to detail presents certain interesting features, both in regard to the probable situation of the lesion and the results of the microscopic examination.

William S., a German, aged fifty-eight, was free from disease until the year 1863 (perhaps 1866), when he was attacked, according to his own statement, with a bubo of the right side. He denied having had a chancre, but shortly after the appearance of the bubo his hair fell out. He continued to be in good health until three years later, when he contracted rheumatism, and at the same time iritis, which was double. These conditions were cured, and we have no further record of the disease until March, 1884, when he suffered from left hemiplegia, the arm and leg being involved in the paralysis, with a cure in twelve weeks. In 1885 he had a second apoplectiform seizure, unaccompanied by palsy, and followed by a rapid restoration to health. In January, 1888, he had left facial palsy, at which time the eye was said to have been inflamed, but there is no definite record as to the character of the inflammation, which evidently was not that which subsequently destroyed his sight. In midsummer of the same year he left the hospital without any evident inflammation of either eye, according to the statement of the trained nurses in attendance.

In January of the next year I made the following note: *Right eye*—Round pupil; light reflex preserved; large, oval, gray optic disc, with dish-like, shallow excavation; arteries as compared with veins smaller than normal; spots of pigment on the anterior capsule of the lens indicating the presence of a former iritis. *Left eye*—Pupil nearly occluded by the former iritis, and preventing any view of the fundus; no evidence of disease of the cornea.

About this time various examinations made by Drs. J. Hendrie Lloyd and Charles K. Mills developed the following facts in regard to this patient's general condition: The left side of the forehead was absolutely smooth, the left angle of the mouth drawn upward, probably due to a spastic condition. There was twitching of the eyelids and muscles of the left side of the face, inability to protrude the tongue, whistle, or dilate the left nostril. The left platysma was weakened, and the masseter and temporal muscles atrophied. The lower jaw could not be protruded to perform grinding movements, sensation was practically lost over the left side of the face, and degeneration reaction present in the muscles supplied by the seventh and fifth nerves. The left arm was paralyzed, with marked atrophy in the extensor and supinator muscles, together with advanced degeneration-reaction and wrist-drop upon this side. The muscles of the left thigh and leg were impaired in power, the extensors and abductors having suffered more than their antagonists, but no atrophy was present, and there was ready response to both faradism and galvanism. Experiments with the tuning-fork seemed to prove the partial deafness which was present to be due to paralysis of the tensor tympani rather than to disease of the auditory nerve.

In March the type of keratitis which caused the destruction of his eye began to develop, and speedily the following conditions obtained, in spite of all treatment: Coarse episcleral injection, perforation of the lower half of the cornea with hernia of the iris, and a filling of the remains of the anterior chamber with blood, and absolute anæsthesia of the cornea and conjunctiva. Lessened intraocular tension.

To sum up, it will be seen that this patient suffered from an atrophic paralysis with degeneration in the left forearm ; a paralysis not complete and not atrophic in the entire left leg ; paralysis of the facial nerve and of the motor branches of the fifth nerve, involving some of the sensory fibres, producing anæsthesia of the left side of the face and of the left eyeball.

Dr. Mills, after an exhaustive study of this case, which has been reported in the *Times and Register* of December 7, 1889, and from which the facts in regard to the examinations other than those of the eye have been quoted, came to the conclusion that this man suffered from a lesion in the anterior horn of the left side, in the seventh cervical segment, and that it reached out to the adjoining segments, and also to the white matter of the crossed pyramidal tracts. The condition of the facial muscles was probably explainable by the presence of a lesion of the same character as that in the cord, destroying the cell groups of both the facial and trigeminal nerves.

The left eye, which caused the man great inconvenience and some pain, and the cornea of which had sloughed, was removed, placed in Mueller's fluid, and submitted to microscopic examination. The following lesions were found :

Below the centre of the cornea there is complete perforation of the cornea, the aperture being occupied by the stump of the prolapsed iris, which is granulating and crowded with numerous inflammatory and large, granular, pigmented cells. This central necrosis, which appears to have begun in the true corneal tissue and spread forward until the slough separated and perforation and prolapse followed, is sharply limited on either side, beyond which the cornea is quite free from inflammatory infiltration. As the periphery of the cornea is reached we find the epithelial layer and overlying conjunctiva much swollen and separated by collections of deeply stained small cells, which have also invaded the corneal tissue, in places massed in pit-like depressions—evidently the earlier stages of abscesses. These lesions do not penetrate the entire thickness of the cornea, and beyond them, passing inward, comparatively healthy tissue remains.

Immediately in the neighborhood of, and surrounding the entrance to, Schlemm's canal, there is a collection of small round cells, apparently extending directly into the lumen of this sinus.

Following the iris from the position of its prolapse toward the ciliary body, this is seen to be swollen, inflamed, and infiltrated with inflammatory cells, a condition which repeats itself in the ciliary body. Of special interest are the vessels in these structures, particularly in the latter. They present one of the forms of obliterating arteritis. Taking as the type a small ciliary arteriole, it reveals in cross-section the following characteristics: An asymmetrically placed lumen contracted out of proportion to the size of the vessel; unusually prominent endothelial plates pushing their way into the central aperture, bounded externally by badly stained fibrous elements that have practically substituted the middle coat, which can still be seen in the periphery as a narrow circle of poorly developed muscular fibres, beyond which the indefinite tissue of the adventitia is apparent. The condition is analogous to that which has been described under the term mesarteritis.

The choroid, retina, and optic nerve are free from disease.

The ciliary nerves, where they pass through the sclerotic coat, were carefully sought out, but do not present, in the carmine sections, any evidence of inflammation or infiltration, and in those stained by Weigert's method any sign of degeneration. Their reaction to these reagents was in all particulars such as is seen in nerves of normal constitution.

The principal appearances as found microscopically are hence: A sharply-defined, central slough of the cornea, separated by nearly normal corneal tissue from a peripherally situated, secondary keratitis, having inflammatory connection with overlying diseased conjunctiva, but bounded below by reasonably healthy cornea; small-celled infiltration surrounding Schlemm's canal; inflamed iris and ciliary body, through which tissues the arterioles present a form of arteritis of the type known as mesarteritis; normal choroid, retina, and optic nerve; and *unaffected ciliary nerves.*

The keratitis present in the eye under consideration, apart from its etiology, is interesting on account of its apparent mode of development. It bears a strong resemblance to the type described by Senftleben (*Virchow's Archiv*, Bd. 65, p. 69), and which he produced in animals in whom the trigeminus had been divided. Under these circumstances this investigator found that the primary affection of the cornea appeared as a necrosis originated by the repeated traumatisms which the eye encountered owing to its anæsthesia. This circumscribed necrosis of the cornea then acted as a seat of inflammatory irritation, and brought into existence a secondary keratitis proceeding from the periphery of the membrane.

The obliterating arteritis evident in the vessels of the ciliary body, in the study of which I have had the advantage of the assistance of Dr. Meigs, presents an inviting opportunity to construct a theory in regard to the etiology of neuro-paralytic keratitis. As, however, was originally pointed out byC. Friedlænder (*Centralblatt f. die med. Wissenschaft.*, 1876, p. 69) arteritis obliterans is found in specimens from clinical and experimental phthisis, in tumors, and in chronic inflammations, especially if accompanied by tuberculosis, an observation which Dr. Meigs has confirmed in his studies of the vessels in various diseased tissues. The existence, then, of this lesion in the present instance is to be explained as the result of a chronic inflammation, and in no sense as a cause of its existence.

The entire absence of disease of the ciliary nerves is a noteworthy fact, and demonstrates that although the nuclei of the fifth pair were affected, there was no degeneration or inflammation in the course of the peripheral distribution of this nerve.

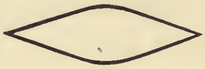
It may be interesting briefly to refer to the various theories which, from time to time, have been advanced to explain the existence of neuro-paralytic keratitis. The earlier experimenters, Magendie, Claude Bernard, and von Graefe, maintained that the destructive changes in the eye were largely due to the section of a trophic nerve. Snellen concluded that the disease was nothing more than a traumatic inflammation provoked by the presence of undetected injuries and foreign bodies. Schiff, noticing that section was followed by paralysis of the nerves of the vessels, together with widening of their calibre and later inflammation, relegated the cause to the nerve itself through a vaso-motor influence, but afterward modified this opinion by stating that a neuro-paralytic hyperæmia was conditioned by the development of an inflammation stirred up by outside influences. The experiments of Samuel, Buettner, Meissner, Eckhard and others gave rise to the tropho-traumatic theory, Meissner especially holding that the inner fibres of the nerve are more important in the trophic influence than any others. According to Buettner and Meissner the division of these fibres deprived the eye of its capacity to resist external influences. Among other theories is that one elaborated by Eberth, in which a mycotic influence was invoked to explain the disease. The pure traumatic theory, developed originally by Snellen, has found many advocates, notably Senftleben and v. Gudden.

The most recent elaborate work upon this subject is by E. v. Hippel (*Archiv f. Ophthalmologie*, Bd. xxxv., 2, p. 217), from whose paper I have abstracted the facts just quoted. This author cannot accept the presence of medially placed trophic fibres in the trigeminus, believes the pure traumatic theory to be untenable, denies the existence of an impaired power of resisting traumatisms on the part of the affected eye, which, however, is more exposed on account of the anæsthesia to desiccation than is the case with a normal one, rejects micro-organisms as etiological factors, and holds that the theory of increased evaporation is sufficient to explain the development of the inflammation, showing experimentally how a moist atmosphere can prevent the disease.

THE INTRODUCTION OF AN "ARTIFICIAL VITREOUS HUMOR" INTO THE SCLERAL CAVITY, IN SIX CASES.

By JOHN MORGAN, of Springfield, Mass.

THE following operations were performed in consequence of having seen a patient at the New York Ophthalmic and Aural Institute, upon whom Dr. Knapp had successfully performed Mules' operation. They were performed under ether anæsthesia. The opening was enlarged horizontally by removing two triangular pieces of the conjunctiva and sclerotic, thus changing a nearly circular opening to one represented by the following diagram,



in all but the first case. In the second case, the pieces cut out were about two lines wide at base, and five in length; in the remaining cases, three lines at base, and five long.

The hemorrhage continued about one hour and a half, and the "artificial vitreous humor" was not introduced until it had completely ceased. A solution of corrosive sublimate, 1 in 5,000, was used as an antiseptic in the first case; a hot saturated solution of boric acid in the second, third, and fourth; and hot water in the fifth and sixth cases. Some experienced no pain, and ate and slept regularly, while others had so little pain or discomfort as not to interfere materially with their eating and sleeping. The wound was closed in all but the first case with six sutures of fine silk, one conjunctival on either side, and four through conjunctiva and sclerotic in the centre. Constitutional disturbance too slight to report.

CASE 1.—Conrad Bersiere, nine years old, *staphyloma of the cornea, and globe much enlarged.* On May 16, 1890, the anterior part of the eye was cut off about one line behind the cornea. After the sclerotic had been freed from its contents, and the bleeding had ceased, a glass sphere of medium size was inserted —which probably occupied less than two thirds the cavity which contained it—and the wound was closed horizontally with fine silk sutures. Hot-water dressings were continually applied for twenty-four hours, after which a compress and bandage, with now and then hot-water dressings while the swelling continued. On the following morning there was some redness and swelling of the upper lid and conjunctiva, which steadily increased until the sixth day, at which time the lids were greatly swollen, and the chemosed conjunctiva prevented their closure. No pain, no discomfort, and no discharge. From this time the swelling gradually subsided, and had nearly disappeared on the thirteenth day, when the dressings were found wet with dark blood, and the wound open at its centre, exposing the glass sphere. On the fifteenth day the wound could not be well closed with a suture, consequently it was enlarged. The sphere and the clot of blood in which it was embedded were removed ; a sphere of smaller size substituted, and the wound closed. On the eighteenth day this glass was visible through an opening of about three *mm* in its longer diameter, which was about one third the size of that on the fifteenth day ; the edges were then freshened, and brought together with an episcleral suture, which in a few days came out and left the aperture as large as before. It was then left to itself, and the patient began to wear an artificial eye. He was seen weekly until August 6th, when the closure was found complete, the sclerotic well contracted upon its artificial contents, providing a support for the artificial eye, which it carries and moves so well that the disfigurement is reduced to a minimum. This is true of all the cases.

CASE 2.—Joseph Thorpe, twenty-eight years old ; during the last week in November, 1889, a fragment of iron entered his eye, and remained there until the day of the operation, June 10th, 1890, when the front part of the eye was cut off immediately behind the iris, the contents of the sclerotic were removed, and their substitute inserted. The two centre sutures were left unclosed for eighteen hours; hot-water dressings for twenty minutes were repeated every three hours, and compress and bandage constituted the treatment for the first twenty-four hours; after that, hot-water dressings for about five minutes, three times a day, and compress and bandage while the swelling continued. Reaction slight. All of the stitches were removed by the eleventh day, when there was an opening of about 3 *mm* at the centre, the edges of which were brought together with an episcleral suture, which proved a failure. Commenced to wear an artificial eye. As there was no apparent change in the size of the opening during the next three weeks, the edges were pared and brought together; this reduced the opening about two thirds; no more interference. Saw the patient weekly, and found wound closed on August 12th. Recovery in nine weeks.

CASE 3.—Katie Donohue, sixteen years old; staphyloma of the cornea. Eye prepared for the reception of the artificial vitreous, as in the preceding case, July 24, 1890, excepting that as already stated the pieces cut out were broader at the base. Hot-water dressings were applied for half an hour at intervals of three hours, and compress and bandage for twenty-four hours; after that, compress and bandage with an occasional use of the hot-water dressings. No pain, no swelling, no discharge, only slight redness of the conjunctiva. Recovered in six days.

CASE 4.—Tillie McGrath, twenty-two years old; staphyloma of the cornea, July 28, 1890; operation same as in case of Katie Donohue, followed by considerable redness and swelling of upper lid and conjunctiva and pain in same side of head. Hot-water dressings, compress, and bandage used as in the other cases. Recovered in eight days.

CASE 5.—Frank Carmody, seven years old; wound of cornea and sclerotic, allowing escape of all the vitreous. About four hours after the injury, August 7, 1890, the eye was prepared for the artificial vitreous, as in the two preceding cases. The eye was dressed with compress and bandage, changed daily for three days, then every other day for four days. No pain, no discharge; swelling of the lids and conjunctiva very slight. August 14th the wound was closed, and began to wear artificial eye.

Case 6.—Wm. D. Pease, forty years old; eye contained foreign body. Operation September 7, 1890, which did not differ from the three preceding cases. Reaction manifested by redness and swelling of upper lid, and conjunctiva, and pain in same side of head. Hot-water dressings, compress, and bandage were used. Recovered in thirteen days.

The patients have all been kept under close observation since the operations, and several of them, including the first, have been seen since Dec. 4th. Some of them have worn their artificial eyes from one to two weeks without removal, and have experienced no discomfort. The conditions appear to be permanent, and the results are satisfactory.

A CASE WHERE REMOVAL OF AN EYE WAS FOLLOWED BY HYSTEROID CONVULSIONS.

By JOHN DUNN, M.D., Richmond, Va.

The patient, a mulatto girl, aged nineteen, came to the clinic of the Richmond Eye, Ear, and Throat Infirmary, September 5, 1890, for treatment of her right eye, the condition of which was as follows: Lids swollen and œdematous. Excessive exophthalmos. Conjunctiva red, swollen, and furnishing a large amount of purulent matter. The cornea had ruptured in the upper one-third of its circumference, and through the rupture appeared a corresponding portion of the iris. The eye was so painful to the touch, and the neuralgic pains through the right side of the head so great that panophthalmitis seemed almost certainly present. The lower two-thirds of the cornea were still transparent in most of their extent. Anterior chamber was obliterated; vision totally gone. The history of the case and the appearance of the eye, though both mother and the girl herself protested in the strongest terms that she had no vaginal discharge, nor had it been possible for her to get the eye inoculated, pointed to gonorrhœal ophthalmia, followed by rupture of the cornea, prolapse of the iris, cellulitis, and most probably panophthalmitis as the causes of the present condition of the eye. The first symptoms of the trouble had appeared about four weeks previously, and the eye had grown steadily worse. The left eye, except a slight congestion of the conjunctiva, was healthy.

As there was no hope of saving the eye, the question arose as to the best method of alleviating the pain and of subduing the inflammation. Enucleation was certainly the best method, but was it safe in the presence of a gonorrhœal process, even though the acute period of the inflammation was over?

Cold antiseptic applications were ordered to be kept constantly to the eye. These lessened in some measure the pain about the eye. On September 8th the patient complained that the vision of the left eye was failing. Examination showed its pupil half dilated, and responding very sluggishly to the light. The conjunctiva showed an increased redness. Patient complained of some pain in this eye. The change in the condition of the right eye was not marked, except that the cornea had become shrivelled and entirely opaque and resembled in appearance a piece of horn. The possibility of a beginning sympathetic involvement of the second eye, even though the ophthalmoscope as yet revealed nothing, determined me to run the risk of a meningeal inflammation and to remove the right eye. The secretion from the conjunctival surfaces was washed off with bichloride solution, and after the removal of the eyeball, the orbital cavity was washed well with the same solution. I then took some iodoform on the end of my forefinger and rubbed it well into the diseased conjunctiva, especially the thickened part on the lids and in the culs-de-sac, using absorbent cotton soaked in bichloride ($\frac{1}{2000}$) as the outer dressing.

Examination of the eye after removal: Passing a probe under the prolapsed iris, it went directly into the vitreous, which I expected to find in a state of advanced inflammation. With a pair of scissors I opened the ball from the prolapse to the optic nerve and thence across to the opposite side. There were no visible disturbances of retina, choroid, or vitreous.

I was unable to see the patient until the following afternoon about two o'clock, when I learned from the mother that, about eight hours after the operation, the patient was taken with vomiting and had had numerous convulsions of the severest kind; that she had called in several of the neighbors to help hold her daughter in bed, and so violent had been her struggles that the bed had broken down under them. I found the patient on a pallet on the floor. She said she had no pain in any part of her body, save in her arm, where a hypodermic of morphine and atropine had been given her prior to the operation. She had no fever. Her skin was moist and warm. Her pulse was 85 and regular. Respiration normal. Pupil of left eye was still somewhat dilated, but responded well to light. There was no discharge from the lids of right eye. And I will remark here that I did not again see any purulent discharge from this eye, to which were kept constantly chlorine applications. I was especially surprised at this, considering the discharge that was constantly present before the operation. And its cessation must be attributed to the rubbing in of the iodoform. The patient had no headache ; answered all questions perfectly rationally, but showed a great desire to sleep, and would fall asleep the instant she was left alone. As patient had not passed her urine for twenty-four hours, I ordered hot applications to the abdomen over the bladder. At 7 P.M. I had to see the patient again, as she was thought to be dying. She had passed her urine soon after the hot cloths had been applied to the abdomen, and had made an attempt to stool. The exertion had probably been too great, for as soon as she was again placed in bed she began to gape, and when I saw her she was gaping about every thirty seconds. The patient would open her mouth to its fullest extent, draw in her breath, and then end the gape with a sigh resembling that of a person very tired. Between the gapes the patient lay perfectly listless. A cup of very strong coffee was ordered to be made and the patient made to drink it. The gaping ceased in a few minutes, and did not return until about 10 o'clock that night, when an attempt was made to change the bed linen which had become soiled. The patient had to exert herself slightly, when the gaping returned. The mother repeated the coffee, and the patient passed a good night. The following afternoon, 11th of September, patient was resting quietly enough, though she complained of some pain beneath the left breast, and was not able to restrain the desire to gape from time to time. The same night patient had, off and on, for two or three hours, convulsions, and with them, opisthotonos, so severe that at times she was resting only on the balls of her heels and the back of her head, and her back so bent that the mother feared it would break. At times the patient would shake her head from side to side, so rapidly and with so much force, that it looked as if she were "trying to shake it off." The convulsions were preceded by attacks of gaping. The afternoon of the 12th the patient was comfortable enough. The only pain of which she had complained during the day was one which would start from the region of her right ovary and would shoot down her thighs. This region was tender on pressure. No fever. Pulse irritable. Right conjunctiva rapidly regaining its normal condition. Irritation symptoms in O.S. have disappeared. The patient says that she remembers the gaping that preceded the convulsions, but of the convulsions themselves and of the opisthotonos she remembers nothing. She remembers shaking her head from side to side, that it was extremely painful, but she could not stop it. Bromidia was ordered, and word was left with the mother to let me know if the attacks returned. I did not see the patient again for ten days, when she came to the clinic. There had been no further return of the hysterical symptoms. The stump had healed.

In looking for a probable origin of the hysterical attacks, I can find none other than the emotional effect upon the higher nervous centres, produced by the idea of the change from the position of belle of her tendom to that of a horrid one-eyed wench. I thought it best not to tell the girl I was going to remove her eye, when I informed her that it was necessary to give her chloroform, for she was run down and much emaciated from the pain she had suffered for four weeks. I told the mother what I was going to do. Thus the girl did not know that her eye had been removed until several hours after the operation, and then, while still weak and sick from the chloroform and still under the influence of morphine, had inferred, from her mother's refusals to answer her questions about her eye, that it had been taken out. Within three weeks after the operation the girl fattened at least fifteen or twenty pounds, so rapidly did her health and strength return when the cause of their deterioration was removed, and it is to this, I think, that must be attributed the fact that the hysterical symptoms disappeared in so short a time.

P.S.—Since the above notes were taken I have learned from the physician who treated the girl before she came to the clinic, that she had a vaginal discharge, most probably gonorrhœal, at the time that her eye trouble began.

A CASE OF INTRACRANIAL NEOPLASM WITH LOCALIZING EYE SYMPTOMS: POSITION OF TUMOR VERIFIED AT AUTOPSY.

By CHARLES A. OLIVER, M.D., of Philadelphia.

(*With a chromo-lithographic perimeter chart.*)

On the 28th of February, 1890, Dr. Francis X. Dercum and the writer met Dr. A. S. Gerhard, the attending physician in consultation in the case of H. N., æt. 39 years, a bridge and wharf builder, who without any history of traumatism, abuse of tobacco or stimuli, or venereal contagion, stated that for two years previously he had suffered with vertigo, headache, and attacks of momentary blindness. In April of 1889 his right foot felt stiff, numb and weak, whilst he repeatedly stumped the toes of his right foot against objects; this persisted until August of the same year. At this time a series of curious motor symptoms of a peculiar nature developed themselves. The right arm was adducted to the trunk, the forearm became flexed upon the arm, the hand was thrown against the forearm, whilst the fingers remained extended, especially the index, the middle, and the ring finger; the seizures holding their maximum for the fraction of a minute and then gradually disappearing.

These attacks became increasingly frequent, until when the patient was first seen they occurred every few minutes. Dr. Dercum, who saw several of these seizures, describes them as distinctly athetosic in character. During the attacks, the right leg would become extended.

When first seen by Dr. Dercum on February 3, 1890, the

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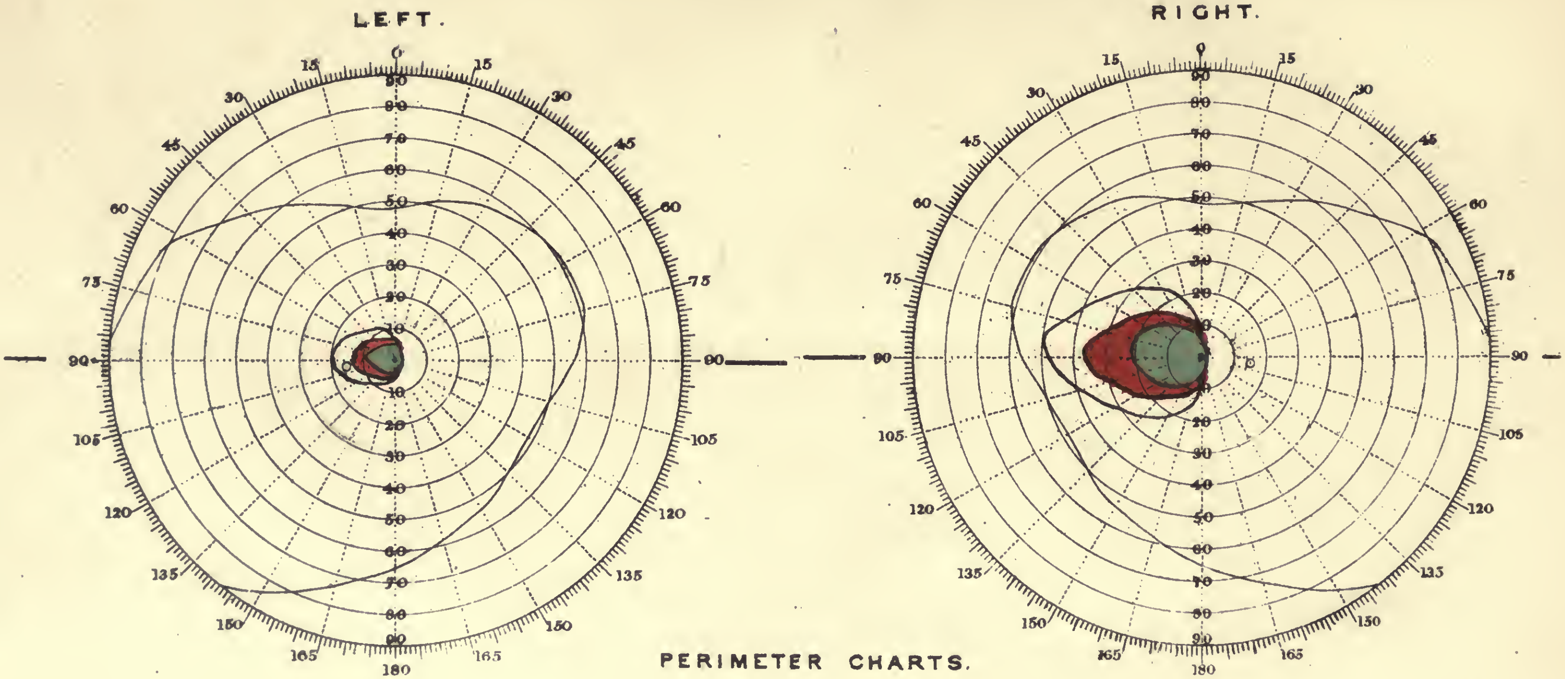
[1] Dr. Francis X. Dercum has presented a short account of this case from a neurological standpoint at the last meeting of the American Neurological Association: the present report which was read before the American Ophthalmological Society, 16th July, 1890, is given from an ophthalmic view only.



LEFT.

RIGHT.

PERIMETER CHARTS.

patient appeared to be a well-built, healthy man with seemingly large, broad muscles. Careful examination, however, showed that the right leg dragged as though spastic. The right grip was weak, and the right patellar tendon reflex was much exaggerated. Right ankle clonus was produced at every examination, whilst knee clonus could be obtained upon the same side at times. No distinct evidences of any mental impairment could be determined. Temperature sense, pressure sense, and muscular sense were all markedly diminished on the right side. Right lateral hemianopsia without any other gross ocular lesion were found.

On the 26th of February, the patient complained of headache associated with marked diminution of sight in the remaining half fields of vision. At this time he became irritable and nervous. Two days later, when first seen by the writer, the following ophthalmic grouping of symptoms were noted :

1. *Acuteness of Direct Vision for Form.* Reduced to $\frac{1}{8}$th. of normal ($V=\frac{5}{40}$) equally on each side, even when properly corrected by minus lenses, thus showing marked disturbance either of some portion of the ocular apparatus which is distributed to both ocular retinæ, or of the ocular tunics themselves.

2. *Direct Vision for Color.* Slightly lowered, especially on the left side; the error manifesting itself for both green and red. This result also shows disturbance either in some part of the ocular apparatus which is distributed to both ocular retinæ or in the ocular tunics themselves, the greater disturbance being on the left side.

3. *Power and Range of Accommodation.* Normal for age and amount and character of error of refraction on each side.

4. *Position of Areas of Visual Fields.* Right lateral hemianopsia, much more marked on the left side.[1]

These conditions of the visual field are extremely important, as they doubly point both by the relative smallness of the left visual fields and the left-sided position of both the remaining areas, towards a disturbance of visual apparatus in the left cerebrum.

5. *Characteristics of Visual Fields.* Feebly negative scotomata marked throughout the remaining green color areas, more pronounced in that of the left eye : this symptom is of immense col-

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[1] The accompanying charts show this very well, the remaining fields of the right eye occupying about one twelfth of their normal areas, whilst those of the left eye are six times smaller.

lateral advantage, as it distinctly shows that the greater amount of irritation is on the left side.

6. *Size of Pupils.* Pupils four millimetres each in horizontal meridian, upon monocular exposure to equal degrees of light stimulus.

7. *Action of Irides.* Wernicke's hemiopic pupillary reaction sign (more properly—hemianopsic pupillary inaction sign[1]) plainly manifest upon both sides; the left iris not so prompt to light-stimulus as its fellow. These two peculiarities of iris behavior—the hemianopsic want of response and the inequality in amount of action taken in conjunction with the fourth observation—serve to show distinctly some disturbance in the sensory-motor arc of the ocular apparatus at the base of the brain in the left optic tract anterior to the corpora quadrigemina and posterior to the optic commissure.

8. *Extraocular Muscles.* With the exception of a slight insufficiency of both interni, extraocular muscle motion intact in every direction.

9. *Fundus Changes.* *a.* Characteristic peculiarities associated with high degrees of myopia equally pronounced in both eyes.

b. Both retinal arteries and veins of the right side, enlarged, tortuous, and apparently carrying dark-colored blood,—a condition which is probably explained by either lymph or blood extravasations into the tissues of the optic nerve or its envelopes.

c. A broad superficial splotch-like hemorrhage, extending over the lower outer quadrant of the right disc, and not seemingly connected with any retinal or choroidal stem. This most curious symptom may be partially explained upon the supposition that the mechanical impediment to the passage of fluid into the intravaginal sheaths of the optic nerve by an intracranial neoplastic formation is greater on the affected side of the brain than on the opposite.[2]

It will be seen that the conditions contained in 2d, 4th, 5th, 7th, and 9th observations, taken together, distinctly point toward a gross left-sided intracranial lesion, situated in such a position as to cause the greatest pressure upon the left optic tract between the corpora quadrigemina and the optic chiasm, a few of the contiguous crossing fibres of the right side being also irritated and somewhat pressed upon; whilst the right-sided hemianæsthesia and the general symptoms upon that side distinctly place the greatest force of the lesion in the region of the left pulvinar.

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[1] Seguin uses the term, "hemiopic pupillary inaction sign." As the term hemiopic has been discontinued, the modified term, as above given, is most probably the best.

[2] *Vide* article on Ophthalmoscopy in vol. iv. of Keating's "Cyclopedia of Diseases of Children." It must be understood, however, that this rule can only be true when the optic-nerve extravasation has been secondary, as it were, to the great increase of cerebral bulk.

During the following three weeks the patient complained more and more of diffuse headaches, until at last he became somnolent and stuporous, dying on the morning of 21st of March.

Post-mortem examination showed a normal calvarium with no special peculiarity of the membranes, except that they were slightly injected and rather dry. The left hemisphere was distinctly bulging, whilst the superficial substance of the brain appeared pale and somewhat anæmic. Upon horizontal section, the left lateral ventricle was found to be shallower than the right, though both were otherwise normal. The left optic thalamus was distinctly indurated and resistant to the touch; its substance being decidedly distended and swollen. Perpendicular section of the hardened mass revealed a neoplasm invading the external portion of the left optic thalamus, as well as the corpus striatum, almost as far as its anterior third. The growth itself was pinkish yellow in color, and was more resistant than the surrounding tissue. The capsule was not invaded. The left optic tract as far as the chiasm was markedly flattened and pressed upon. The right brain was seemingly untouched and unharmed.

Microscopic section of the tumor revealed a glioma with commencing sarcomatous degeneration. Study of the optic tracts, chiasm, and optic nerves gave negative results.

THE LIGHT-STREAK AS SEEN UPON THE CENTRE OF THE RETINAL VESSELS, DUE TO REFLECTION, REFRACTION, OR TO BOTH?

By ACHILLES E. DAVIS, A.M., M.D.,

SENIOR ASS'T HOUSE SURGEON, MANHATTAN EYE AND EAR HOSPITAL, N. Y.

(*With Drawing.*)

THE light-streak seen upon the centre of the retinal vessels, and its causes, has been a fruitful subject for discussion since Loring's theory, in 1870, in contradiction to the accepted theory of its cause up to that time.

In 1853, Van Trigt[1] first advanced the theory of the production of the light-streak, as seen upon the retinal vessels, by the *reflection* from the anterior surface of the vessel-wall back into the observer's eye, of those rays of light from the ophthalmoscopic mirror, which fell directly on the summit of the retinal vessels. In this theory, shortly afterwards, he was sustained by Jaeger,[2] who accepted it *in toto* at the time, but in after years (1869, in his last book[3]), though adhering to his former view of the subject, ascribed the phenomenon to a reflection of light from the anterior surface of the *blood-column*, rather than to a reflection from the anterior surface of the *vessel-wall*, as he formerly maintained. Mauthner, in his work, 1867, also accepted the theory of Van Trigt, claiming that the light-streak was due to reflection of light from the anterior surface of the vessel-wall. In fact, the theory of *reflection* was accepted by the profession in general, and was not questioned until 1870, seventeen years after its first advancement by Van Trigt; when Loring, in his celebrated article[1] on this subject, took an exactly opposite view of the question, and advanced the theory of *refraction* of light as the chief cause in the production of the light-streak.

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[1] Dissertat. ophthal. inauguralis De speculo oculi.

[2] "Ergebnisse d. Untersuchung d. mensch. Aug. m. d. Augenspiegel," 1854.

[3] "Ophthalmo. Hand. Atlas," 1869.

He attributed the presence of the light-streak, as seen upon the centre of the retinal vessels, not to the *reflection* of light from the anterior surface of the vessel-wall, or from the anterior surface of the blood-column, but to the *refraction* of those rays of light falling on the summit of the vessel, penetrating it and the blood-column, these being reflected slightly by the posterior wall of the vessel, but chiefly by the underlying tissues, back through the vessel and blood-column into the eye of the observer, the refractive action of the blood-column condensing the light as it passes through it both ways, just as a bi-convex lens, thus causing the light-streak. (See drawing on next page.)

In proof of this view, he not only cited many facts, but performed a most unique and simple experiment, which I will give briefly for the benefit of those who have not read of it, and as I make a similar experiment, differing in one essential point only, that is, to use the actual blood-column where he used a solution of carmine and water.

In his experiment he took two cylindric boxes, about three inches long and one inch in diameter each, fastened them together longitudinally; at the bottom of one, which we shall call 1, he placed a reflecting surface, an ordinary ophthalmoscopic mirror, and at the bottom of the other, which we shall designate 2, he placed a non-reflecting surface. Just in front of the reflecting surface in the one case and the non-reflecting surface in the other, in a transverse direction, he passed a very thin glass tube about a line in diameter, containing a solution of carmine and water.

In a rough way he let this represent the human eye, the mirror representing the retina, and the tube with carmine solution a retinal vessel.

Casting the light down these cylinders with an ophthalmoscope, just as in the examination of an eye, he obtained in the cylinder with the reflecting surface—the mirror at the bottom—a light-streak on the tube containing the carmine solution, just like that obtained on the retinal vessels of the eye; whereas when he cast the light down the cylinder with the non-reflecting surface at bottom, he obtained a diffuse red light, which extended across the entire diameter of the tube containing the carmine solution, and not a light-streak, as in the other cylinder; thus showing that where the reflecting surface is wanting behind the tube in the one case, and behind the retinal vessel in the other, as in detachment of retina, there also is the light-streak wanting; and, *vice versa*, where there is a reflecting surface behind the tube or retinal vessel to reflect the light back through them a second time, there the light-streak is present. Furthermore, if the light-streak depended *solely* on the reflection of light from the anterior surface of the tube or vessel, and not on the refraction of light passing through it and reflected from the underlying tissues, then the presence of a reflecting or non-reflecting surface *behind* the tube or vessel would be immaterial, according to Van Trigt's theory, in its production.

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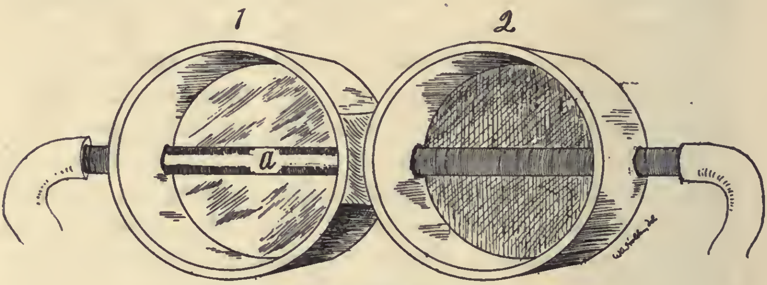
[1] *Trans. Amer. Ophthal. Soc.*



1. Cylinder with reflecting surface at bottom.
2. Cylinder with non-reflecting surface at bottom.
a. Hole in ophthalmoscopic mirror.

This instrument was kindly made for me by Messrs. Georgen & Hann, opticians, 54 E. 23d Street.

But we do not find this to be so; in fact, we find that we obtain a light-streak only in that instance where we have a reflecting surface beyond the tube or vessel, thus showing that the light-streak depends upon something more than simple reflection from the anterior surface of the tube, for if it did not, then we should get it in the cylinder with a non-reflecting surface at the bottom, as they are seen, so far as they themselves are concerned, under precisely the same conditions.

The analogy of the instrument used by Loring to the human eye, so far as physics are concerned, is exact, and, consequently, what is true of the one is also true of the other.

But as clearly and completely as his experiment seemed to prove the cause of the presence of the light-streak on the retinal vessels, it still left the solution of the question in doubt, and by many writers the truth of his theory was stoutly denied. Notably among those who opposed the theory of *refraction* was Schneller,[1] who maintained the theory of *reflection* as advanced by Van Trigt in 1853, and brought forth many explanations to sustain his objections, but all of which were untenable as afterwards shown by Loring.

H. Knapp[2] has this to say about Schneller's article against Loring's theory: "Schneller's experiments and statements are, in part at least, as far as can be judged without repeating them, correctly refuted by Loring."

Prof. F. C. Donders was another to oppose Loring's theory and to sustain Van Trigt's, and, as he says, for the following reasons (which I give in full, as I expect to refute them):

"1. The blood-cylinder in the vessels of warm-blooded animals (Becker examined frogs only) is non-transparent; consider that light has to traverse this cylinder twice in order to form, according to Loring, a light-streak by refraction.

"2. The light, after passing twice through the blood-column and undergoing the numerous reflections and refractions on both sides of the blood corpuscles, must be totally diffuse and unfit to produce an image.

"3. Such an image would be situated in front of the wall of the vessel, not behind it.

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[1] Graefe's *Arch. of Ophthal.*, xviii., 1, pp. 113–126.

[2] Knapp's ARCH. OF OPH. AND OTOL., vol. iv., No. 1, p. 147.

"4. The light-streak remains unchanged when an artery runs directly over a wide, darker vein which cannot send back the same kind of light through the artery. Furthermore, the tapetum of animals and morbid changes, by which the fundus oculi becomes lighter, have no material influence on the presence of the light-streak."

To objection No. 1 (and this is the one he lays most stress upon), "that the blood-cylinder is non-transparent in *warm-blooded* animals," I can give positive proof as to its invalidity by the following very simple experiment. To perform the experiment I used an instrument similar to that used by Loring in his experiment which I have described above, with this addition to it, to wit: I attached to either end of the very thin glass tube that extends transversely across the two cylindric boxes two small rubber tubes, letting one of these tubes extend into a basin of water, while the other tube had a cone-shaped glass tube fastened in its end—for what purpose I will mention a little later. To obtain a column of blood from a *warm-blooded* animal I made a vivisection on a cat, first etherizing it, then dissecting down upon one of the carotid arteries, and making a small opening into it, introduced the cone-shaped glass tube above mentioned into this opening, which conducted the blood to the small rubber tube which was attached to it, and this in turn conducted it to the small glass tube (1‴ in diameter) which ran through the instrument, and thence through the other rubber tube into the basin of water. The reason for conducting the blood to a basin of water was to keep the flow of blood for some minutes. In other words, I made a simple transfusion of blood from the cat to the basin of water in the same manner exactly as we transfuse blood from one animal to another, or from an animal to man. With the proper assistance this is easily done.

Now, with every thing in readiness, the column of blood flowing through the thin glass tube just in front of the mirror in cylinder 1, in the one case, and the non-reflecting surface in the other cylinder, 2, I cast the light down the cylinders from an ophthalmoscope just as in the examination of an eye, and obtained exactly the same phenomena as

Loring when experimenting with a solution of carmine and water—that is, in the cylinder with a mirror at the bottom, a light-streak along the centre of the tube containing the blood-column, this light-streak being limited, both above and below, by a darker red border, but disappearing altogether from the tube when the tube runs in front of the hole in the mirror (this last fact I mention because I will have occasion again to call attention to it). Secondly, on looking down cylinder 2, we do not get any light-streak at all on tube also containing the blood-column as in cylinder 1, but a diffuse red light extending across the entire diameter of the tube.

But why should we obtain two such opposite results in looking down the cylinders, *e.g.*, a light-streak on the tube in one, a diffuse red light on the tube in the other? Evidently not because of any difference in the tube in the cylinders, for it was one and the same tube; certainly not to any difference in the blood, for it was one and the same blood; it must be, therefore, due to some condition external to the tube, and this condition, in this instance, is a difference in the surfaces beyond, the one a reflecting, the other a non-reflecting one, thus showing that the light-streak depends upon the character of the surface beyond the tube. However, according to Van Trigt's and Jaeger's theory of the production of the light-streak, that is, to a simple reflection of light from the anterior surface of the vessel wall or its contents, the surfaces beyond the vessel or tube would have no influence in the production of the light-streak one way or the other. But we find that it does, and in this very instance, for "if," as Loring says, "the light-streak seen in 1 was simply due to a reflection from the wall of the tube, or its contents, then we ought to get it also in 2, as the tube is one and the same as that in 1, and is seen, so far as it is itself concerned, under precisely the same conditions."

Furthermore, that the light-streak does depend upon a reflecting surface beyond the tube in order that light may be reflected back through the tube and acted upon by the refractive power of its contents, and not to a simple reflection of light from the anterior surface of the tube or its contents, or even the posterior wall of the tube, is shown from the fact that just where the glass tube passes in front of the hole in the mirror—that is, when the reflecting surface, beyond the tube is wanting—there likewise is the light-streak wanting (see drawing, *a*, No. 1). "This is due," as Loring says, in his experiment with the carmine solution with a similar result, "evidently to the fact that just where the hole is, the light after having passed through the tube does not strike upon the mirror, but passes through to the bottom of the box beyond, which being of a non-reflecting nature does not permit sufficient light to come back to the observer's eye to produce the light-streak."

Now, therefore, having gotten *in every detail* the same phenomena with the *actual warm blood-column* that Loring did with the carmine solution, proves: *first*, *that the blood-column in warm-blooded animals is transparent; secondly*, *that it does transmit light*, *at the same time exerting a refractive action upon it; thirdly*, *that it does produce well-defined images*, *e.g.*, the light-streak on the tube in cylinder 1, and on the retinal vessels in the eye.

And the fact that it does produce a well-defined image nullifies Donders' second objection to Loring's theory, to wit: "the light, after passing twice through the blood-column and undergoing the numerous reflections and refractions on both sides of the blood corpuscles, must be totally diffuse and unfit to produce an image."

To further establish the fact that the light-streak is *due* to the refractive action on light of the blood-column, and to transmitted light; and at the same time that the blood-column in warm-blooded animals is transparent and capable of producing well defined images by its refractive power on light, I performed the following experiment. A vivisection was made upon a cat, as before, cutting down on one of the carotid arteries in the neck, transfusing the blood from this through the same tube that I used in the previous experiment. This time I placed the very fine glass tube, with the blood flowing through it, under the microscope, using a very weak objective, also a weak eye-piece. Here I obtained a similar result to that obtained by Becker,[1] in his experiments with the mesentery of frogs under the microscope, to wit: a light-streak on the summit of the tube, similar to that upon the summit of the retinal vessels of the eye. Therefore it is evident, and according to a law of physics, which can be disputed by none, that the blood-column in *warm-blooded* animals, as well as in cold-blooded (Becker on frogs), is transparent, does exert a refractive action upon light, and is capable of producing well-defined images. That the light-streak in my last experiment was produced by transmitted light, refracted by the blood-column, and not by reflected light from the anterior surface of the blood-column, cannot but be evident to the least observing. Consequently, this fact being admitted, it not only makes the theory of refraction as the cause of the light-streak, as first advanced by Loring, in 1870, wholly tenable, but establishes it as an indisputable fact, sustained by optical and physical laws.

The other two objections—Nos. 3 and 4—of Donders, to Loring's explanation of the cause of the light-streak, can easily be disposed of.

No. 3. "Such an image" (speaking of the light-streak) "would be situated in front of the wall of the vessel, not behind it." By which objection he upsets his own theory, as advanced by Van Trigt; for if, as they claim, the light-streak be due to a reflection of light from the anterior surface of the blood-column, then the image would, being reflected from the anterior surface of the blood-column, necessarily be behind, and not in front of the vessel-wall. Whereas, according to the theory of refraction, it would be in front of the vessel-wall; and such is really the case, as the blood-column acts as a bi-convex cylinder, as we have shown above, and the image it produces must, according to known optical laws, be in front, not within or behind it. So much for No. 3.

Objection No. 4. "The light-streak remains unchanged when an artery runs directly over a wide, darker vein, which cannot send back the same kind of light through the artery, etc." This objection, I think, was answered by Loring himself, when he gave in proof the example of the light-streak disappearing from the retinal vessels in detachment of the retina, ascribing the cause for its disappearance here to the diffusion of light by the fluid beneath the retina, for the light-streak remains unchanged on the vessels, up to the very edge of the detachment, but disappears immediately, as soon as the vessel runs upon the detached portion. Therefore, if the light-streak was due to the reflection of light from the anterior surface of the blood column, then it ought not to disappear from the vessel upon the detached portion, for the underlying fluid would have nothing to do with it in that case.

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[1] Graefe's *Arch. of Ophthal.*, xviii., p. 280.

To add to this already convincing evidence in favor of refraction are two cases in its support, given by Noyes[1] in his late text-book on "Diseases of the Eye." In one of the cases (I give in Noyes' own words) "there was an effusion of blood beneath the choroid, which made a dark patch. This was crossed by a vein, on which no light-streak was present, while it traversed this dark surface, but when situated upon the normal choroid, the usual streak was present. As the blood-patch became absorbed, and a white scleral surface came to view, which was caused by rupture of the choroid, not only did the vessel recover its usual light-streak, but this became much more decided than upon the adjacent portions of the vessel. A second case bearing upon this point was one of extreme colloid deposit upon the choroid, having all the brilliancy of the most marked patches of fatty degeneration, as found in albuminuric retinitis. This glittering surface was about two discs long and one disc wide, and was behind one of the transverse retinal arteries. As the artery crossed the spot, the whole vessel was a bright ribbon of light, the central streak being intensified and widened, so as to equal the diameter of the vessel. On either side of this spot the artery had the usual appearance."

With such confirmatory evidence as this, and coming from such authority, I think we can safely let the question rest; though it is in opposition to such eminent authority as Van Trigt, Jaeger, Donders.

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[1] Noyes' "Text-Book on Diseases of the Eye," p. 50.

A CYSTIC TUMOR OF THE LACHRYMAL GLAND.

By MATTHIAS L. FOSTER, M.D.,

ASSISTANT SURGEON MANHATTAN EYE AND EAR HOSPITAL, LECTURER AT THE NEW YORK POLYCLINIC, OPHTHALMOLOGIST TO THE LADIES' DEBORAH NURSERY AND CHILD'S PROTECTORY AND TO THE UNITED HEBREW CHARITIES, LECTURER ON DISEASES OF THE EYE AND EAR AT THE H. W. BISHOP TRAINING SCHOOL FOR NURSES.

Read before the Section in Ophthalmology, Academy of Medicine, New York City, Oct. 20, 1890.

A lady, aged forty-five years, came to me in February, 1890, to seek relief from a tumor which had displaced her eye and was causing considerable pain. An operation had been performed six years before, by a local physician, who evacuated a quantity of watery fluid through an incision in the upper lid. The tumor immediately recurred and slowly increased in size, and for two weeks previous to her consultation with me the pain had been excessive.

On examination I found the upper portion of the right orbit occupied by a soft, fluctuating, apparently lobulated tumor, measuring two by one and a half inches. The eye was flattened from above downward, and pressed forward and downward upon the cheek so far that the lids did not quite close over it. The cornea was hazy, the pupil contracted, vision = fingers at four feet. An ophthalmoscopic examination was attempted, but was found to be very difficult and had to be abandoned on account of the nervous condition of the patient. On eversion of the lids the bluish appearance described as characteristic of dacryops was not to be seen. The patient claimed that tears came from the eye when she cried.

The same afternoon the patient was placed under the influence of ether. After the eyelids and adjacent skin had been thoroughly cleansed with soap and water and a solution of corrosive sublimate, an incision two inches long was made over the middle of the tumor, about a quarter of an inch below the supraorbital ridge, parallel to the cicatrix which resulted from the former operation. An attempt to dissect out the cyst in its entirety had to be abandoned as impracticable, because a large part of the wall of the main cyst was firmly adherent to the periosteum covering the orbital plate of the frontal bone. This main cyst extended backward an inch and a half from the supraorbital ridge, and contained at least ℥ ss. of clear, straw-colored fluid. It may give a better idea of the size of this cavity if I state that I passed the two distal phalanges of my forefinger into the orbit, and while its palmar surface pressed upon the orbital plate, its dorsal surface did not touch the eyeball, which was lying beneath it.

Two smaller cysts were found, one beneath the angular process of the frontal bone, behind and above the lachrymal gland ; the other almost wholly in the lid. The lachrymal gland was held in the midst of these three cysts and was divided irregularly into several portions, but I was unable to determine whether this division was the result of wounds received during the former operation or of degeneration due to the pressure of the cysts.

The cyst walls were separated as thoroughly as possible from the neighboring tissues until a mass was formed of cyst wall and lachrymal gland, having a pedicle attached to the conjunctiva near the retrotarsal fold, close to which it was divided. It was then believed by all who witnessed the operation that no trace of the lachrymal gland had been left in the orbit.

The wound was thoroughly cleansed with a boro-salicylic solution, and closed with three silk sutures. The eye was then replaced in its normal position in the orbit, and a moderate pressure was applied by means of a sublimate dressing.

The wound healed readily except near its outer angle, where a sinus persisted for about six weeks. Shortly after its closure evidence of a re-collection of fluid appeared in the inner part of the lid.

On the 8th of July I found that this collection had increased considerably and was causing some pain, so I passed a Graefe knife upward and backward through the retrotarsal fold into the cyst and drew off a quantity of fluid. Since then the patient has been quite comfortable, suffers no pain, and is able to read rather coarse newspaper print. She has a reasonable supply of tears in the eye, and claims to have considerable epiphora when exposed to a bright light or a strong wind.

Tumors of the lachrymal gland are so rare that very little space is accorded them in the majority of the text-books, and mention of them in general literature is not common. Among these tumors the most frequently met with perhaps is that known as dacryops, a condition closely analogous to ranula, caused by the closure of one of the ducts leading from the gland. This closure causes the retention of the tears in a sac, formed at first by the walls of the duct, later by the surrounding connective tissue, which becomes more and more distended as the lachrymal gland continues to add to the imprisoned secretion. This may continue until the eyeball becomes pressed downward and forward upon the cheek, giving way to the cystic tumor which occupies the upper part of the orbit. When this is the condition, a bluish, translucent, cyst-like tumor, covered only by the conjunctiva, may be seen on eversion of the lid.

The best mode of treatment for this affection seems to be a little doubtful. In an uncomplicated case puncture of the sac through the conjunctiva and maintenance of the patency of the opening so formed would appear to me the preferable method. Simple evacuation of the cyst through an incision in the lid with or without cauterization of the sac, or an attempt to dissect out the cyst without removal of the lachrymal gland cannot be productive of good. The excretory ducts will be more firmly and completely closed than before by the formation of cicatricial tissue, while the gland itself will retain its normal activity, so that either a permanent external fistula will result or the cyst will rapidly recur, and the final condition of the patient will be worse than the first.

If the gland be removed with the cyst we may reasonably expect no recurrence and no fistula. Still, although there will be sufficient mucous secretion to keep the conjunctiva moist after its removal, the lachrymal gland has its functions and should not be removed so long as there is a hope of successfully treating the cyst in another manner.

In cases which have become complicated by previous attempts at surgical interference or otherwise it may not be best to delay the radical operation. In my own case I believe that puncture would have utterly failed at the time I first saw the patient. It might have evacuated one or possibly two of the cysts and given temporary relief, but I feel certain that the third cyst which extended up into the lachrymal fossa would not have been touched, would have caused a quick recurrence of the symptoms and would have necessitated the radical operation at a later date.

It is difficult for me to account for the recurrence which really did take place except by the theory that a small accessory gland was present and overlooked in the wall of the cavity. The dissection was conducted in the most careful manner. The lachrymal gland was dislocated into the cavity formed by the cysts, and was separated by them from the walls on every side. The cysts were separated from the muscular tissue before they were opened, and were afterwards scraped as thoroughly as possible from the bone. In this manner a mass was formed which was attached nowhere except to the conjunctiva at or near the mouths of the excretory ducts, and no portion of the mass was left when this pedicle was divided. If the mass had been cut through and a portion left when the incision through the pedicle was made, it would have certainly been detected a moment later when I examined the stump. It is possible that a portion of the gland may have been cut off during the previous operation, become widely separated from the rest by means of the growth of the cyst, and been left unnoticed in the wall of the cavity. A portion so cut off would act as an accessory gland, and could not readily be differentiated from it. Its presence in this case is hardly to be regretted so long as an opening through the conjunctiva is patent, as it seems to be performing the functions of a lachrymal gland as well as can be desired.

PUNCTURED WOUND OF THE SCLERA; RECOVERY WITH GOOD VISION.

By W. E. BAXTER, M.D.,

SURGEON TO EASTERN MAINE EYE AND EAR INFIRMARY, BANGOR, ME.

Mr. W. age twenty-seven, American engineer, was brought to my office October 7, 1890, by Dr. Edmunds of this city. He states that half an hour before I saw him he was struck on the outer part of the right eye by a piece of broken glass from the water-gauge from his engine. On putting his hand to his eye he found, as he states it, " part of his eye running out"; he then put his handkerchief over his eye, and held it there until he got to my office.

An examination showed a large vertical wound in the eyeball, 2 *mm* from the outer border of the cornea, 8 *mm* in length and 3 *mm* in width, through the conjunctiva and sclera, large enough to admit the end of an ivory-handled eye instrument ; through this wound the vitreous was protruding; there was a loss of scleral tissue at the middle part of the wound. The anterior chamber was partly filled with blood, and the cornea collapsed.

After cleansing the wound, the conjunctiva was dissected from the sclera for some distance around the wound ; the sclera was brought together with the silk sutures, and the conjunctiva brought together, so sealing the wound in spite of the loss of scleral tissue. Having no catgut fine enough for the sutures, silk had to be used, but no trouble was found in removing these through the conjunctiva.

Four days from this time the stitches were removed, perfect union having taken place. The anterior chamber was cleared from blood. The vitreous was somewhat hazy, but the patient could distinguish objects, the color of one's eyes, etc. At the end of two weeks the eye was free from irritation, and the vision $\frac{6}{12}$ and improving ; since then the patient has not been seen.

NOTE CONCERNING ASTIGMATIC BIFOCAL LENSES.

By GEORGE M. GOULD, Philadelphia, Pa.

CONCERNING the opinion of Mr. Percival, that the bifocal lenses are not suitable for astigmatic patients, I would say that I never make any difference in such cases, and that the great majority of my presbyopic patients are astigmatic. I have never heard a complaint from them, and cannot understand why our esteemed colleague finds the lenses unsuitable. I have patients wearing sphero-cylinders, both plus and minus, at all axes, and sometimes the astigmatism running as high as six dioptries, and with perfect satisfaction. Thinking the explanation may consist in the method of manufacture, I have sent Mr. Percival a sample of a pair of lenses that has been worn for six months, of the following refraction:

Distance { O. D., — sph. 2.25 D., ⊃ — cyl. 3.00 D. ax. 95°.
O. S .,— sph. 5.00 D., ⊃ — cyl. 1.25 D. ax. 90°.

Reading { O. D., + sph. 0.50 D., ⊃ — cyl. 3.00 D. ax. 95°.
O. S., — sph. 3.00 D., ⊃ — cyl. 1.25 D. ax. 90°.

ON SOME OPHTHALMIC SEQUELÆ OF THE INFLUENZA EPIDEMIC.

By W. M. BEAUMONT,

SURGEON TO THE BATH EYE INFIRMARY, ENGLAND.

AFTER so wide-spread an epidemic as that of last winter, one from which no nation, class, age, or sex seems to have been exempt, and one in which the after depression was so marked a feature, it is not surprising to find that, though the disease has now departed, it has left behind traces of its havoc. All sorts and conditions of aches and pains, forming a heterogeneous mass of nondescript symptoms, are dated from an attack of influenza. In many cases there certainly is, whilst in others there certainly is not, a link of cause and effect, and yet again in a large number of instances one is left in doubt as to whether the attack of influenza is a *vera causa* of, or a mere precurrent casualty to, the subsequent ailment. No class of sequelæ seems to be more frequent than the ophthalmic; and no variety of eye-disease, if we may believe our patients, but what may follow an attack of influenza. I have seen patients, the victims of senile cataracts, who accounted for their blindness by such an attack, although on being questioned they have granted that the sight was not good previous to the illness. In the following list of cases I have only admitted such as seemed to be so closely coincident with the attack of influenza that there might be a fair presumption, or at least the possibility, of a relationship of cause and effect; it must not, however, be forgotten that ophthalmic sequelæ often follow an illness, although that illness is not one that would be expected to interfere with the function of the eye. As far as my limited experience goes it seems, as has been pointed out by Fage,[1] that the later attacks of influenza, that is, those which occurred when the epidemic was on the wane, were more prolific in producing ophthalmic sequelæ than those attacks which occurred at the beginning of the outbreak. I have notes of twenty-two cases, in all of which some form of eye-disease accompanied or supervened upon an attack of influenza, and of these cases I append the following brief notes:

CASE 1. *Blepharitis and Hypermetropia.*—A woman, æt. 47, was laid up with influenza in March, and as soon as she got about again, which was in the first week of April, the lids became red and painful and sewing was impossible. She did not, however, put herself under treatment until May, when a bicarbonate of soda lotion and + 2 D glasses quickly cured her.

CASE 2. *Hypopyon-Keratitis.*—A woman, æt. 66, took to her bed on account of influenza in February, and whilst ill with it the left eye became "inflamed" and very painful. She came to the Bath Eye Infirmary in April, when the anterior chamber was half full of pus and vision barely p. l. Paracentesis was performed, but it was a month later before the eye became quiescent.

CASE 3. *Paralysis of Accommodation.*—A man, æt. 57, had an attack of influenza in February. Whilst convalescent he discovered that he could not see distinctly either at a distance or near, but an attack of diarrhœa coming on, which lasted nearly four weeks, he took very little notice of the defect of vision. When he came to the Eye Infirmary on May 15th the pupils were found to be very sluggish to light and inactive to accommodation (B E $\frac{6}{60} + 3 = \frac{6}{6}$ p. No. 19 J. + 8 = 1 J). There was no history of sore-throat or syphilis, and vision was the same under atropine. He improved a little during the next two months, and was then lost sight of.

CASE 4. *Blepharitis, Hordeola, and Mixed Astigmatism.*—A girl, æt. 9, was laid up with influenza before Christmas, and a week after she recovered the eyelids became red, sore, and covered with "wisps." She remained in much the same condition until May, when a moderate degree of mixed astigmatism was corrected and the lids got well. She had never had any thing the matter with the lids before the attack of influenza.

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[1] Complications oculaires de la Grippe.—*Archives d'Ophtalmologie*, t. x., p. 146.

CASE 5. *Muscular Insufficiency, Heterophoria.*—A lady, æt. 65, had an attack of influenza in March and a relapse in April, and this again was followed by an attack of bronchitis in June. Previous to her illnesses she read easily with + 2 D, but she was not able to use her eyes comfortably afterwards. In September she was found to read, with + 3 D for the R and + 2 D for the L, J. 1 at 33 *cm*, when the eyes were used separately, but with binocular vision lenses were of very little assistance to her as the type became confused and the eyes tired at once. On making her converge it was found that a quivering of the eyes resulted, and when either eye was abducted extremely the same happened. Prisms were of no assistance to her, but under ammoniated citrate of iron she eventually recovered.

CASE 6. *Chronic Conjunctivitis.*—A man, æt. 37, was confined to the house with influenza for three weeks in January. On the first day that he went outside after the attack he "caught cold in the eyes," and was in consequence obliged to keep in-doors for a fortnight. The disease then ran a chronic course for three months, after which he came to the Bath Eye Infirmary (May). The conjunctivæ, both ocular and palpebral, were found to be inflamed, and there was a good deal of muco-purulent discharge as well as pain, this latter being referred to the back of the eyes. The iris was not affected, and his vision was normal.

CASE 7. *Muscæ.*—An anæmic girl of 21, who said her sight had always been weak, was ill with influenza in January, and ever since she had been annoyed by muscæ. Her vision was good, the refraction emmetropic, and she improved rapidly with iron and arsenic.

CASE 8. *Catarrhal Conjunctivitis.*—A woman æt. 38; never had any thing the matter with her eyes until she was laid up with influenza last February. A week after its onset the corners of her eyes, she said, became painful and inflamed. There was no error of refraction. $V = \frac{6}{6}$.

CASE 9. *Internal Strabismus, Diplopia.*—A woman, æt. 49, was laid up with influenza and sore-throat in January. In April she came to the Eye Infirmary complaining of homonymous diplopia. The R was found to be slightly converged. Vision, $\frac{6}{6}$ with each eye. The diplopia was neutralized by a prism of 5°, base outwards (Landolt's series). The optic discs were normal, T n.

With + 2.5 D she read J 1 easily. After two months' treatment with iron and strychnine she quite recovered.

CASE 10. *Paralysis of the Accommodation.*—A woman, æt. 40, had a severe attack of influenza at Christmas, followed by a relapse, or, perhaps more correctly, a second attack, in February, after which she noticed that her sight was dim. There was a history of syphilis. In June the left eye was found to be amblyopic. (It had been operated upon for strabismus in childhood.) R E, $\frac{6}{60}$ + 7 D = $\frac{6}{8}$ and J 16. With + 10 J 1. No mydriasis. The pupils act to light but not to accommodation.

CASE 11. *Glaucoma.*—A lady, æt. about 60, was laid up with influenza about Christmas, and her eyes at the same time became "irritable." They did not get any better when she convalesced from the attack of influenza, and when seen in April she was in the following condition: The R pupil was dilated, the media muddy, T + 2, and she could only see shadows. The L pupil was not dilated, but was extremely sluggish; she saw halos round the candle; the anterior chamber hardly existed; T + 1 and vision $\frac{6}{36}$. By the ophthalmoscope the disc was seen to be cupped. Double iridectomy was followed by no improvement in the R, but in the L vision became $\frac{6}{12}$.

CASE 12. *Conjunctivitis and Hypermetropia.*—A farmer, æt. 19, was confined to bed with influenza in March. In June he came complaining that he had had visual troubles ever since. His refraction was found to be H. = + 1 D, and there was in addition some irritable follicular conjunctivitis.

CASE 13. *Episcleritis.*—A man, æt. 61, had influenza in March, and at the same time pain and lachrymation in the R eye. When seen in May there was a patch of episcleritis just external to the corneal limbus. His refraction was emmetropic.

CASE 14. *Asthenopia, Hypermetropia.*—A lady, æt. 25, was laid up with influenza last February, after which she was unable to follow her employment as a teacher in consequence of headache and blurring when she attempted to read. In July she was found to have Ht. 3.5 D. With lenses + 1.5 she was able to resume work at once.

CASE 15. *Conjunctivitis and Hypermetropia.*—A woman, æt. 28, was attacked by influenza last Christmas. This was followed by photophobia and conjunctivitis. She applied for treatment in April, and was quickly relieved by boracic fomentations and + 1 D spectacles for near vision.

Case 16. *Phlyctenular Conjunctivitis and Myopia.*—A woman, æt. 45, was laid up with influenza last February. She came to the Eye Infirmary in April, complaining of photophobia due to phlyctenular conjunctivitis. She was myopic (R — 1 D, L — 2.5 D), but had never worn glasses. Under treatment she slowly convalesced during the next five weeks, due attention being paid to the error of refraction.

Case 17. *Blepharitis, Hypermetropia.*—A lady, æt. 44, suffered from typhoid four years ago ; since then the eyes have been sore and red, and occasionally they have discharged matter. Last January she was laid up with influenza, and since then the eyes have been very much worse, and she has had difficulty in reading small print. When seen in June there was a good deal of conjunctivitis and lachrymation ; her refraction was hypermetropic, 1 D. Boracic lotion and + 1 D glasses quickly relieved her symptoms, and in July she was quite well.

Case 18. *Asthenopia, Hypermetropia.*—A lady's maid, æt. 21, had to give up work in the last week of January in consequence of an attack of influenza. She came to the Eye Infirmary in April, saying she had not been able to sew since her illness. On examination there was found to be 1.75 D of hypermetropia. With + 1 D she was able to resume work at once.

Case 19. *Asthenopia, Hypermetropia.*—A gentleman, æt. 43, was laid up with influenza in February, followed by aching in the balls of the eyes whenever he attempted to read. He had never suffered thus before and he had never worn glasses. The correction of his Ht. (R + 2, L + 3) relieved all the symptoms.

Case 20. *Blepharospasm, Anisometropia.*—A young lady, æt. 26, was laid up with influenza in February, and as soon as convalescence was established she found that she could not use her eyes for near work in consequence of the continuous blinking of the lids. There was no disease of the teeth. Shower-baths had been tried in vain. When seen in September, seven months after the blepharospasm, her refraction was found to be, R, Ht. 1 D ; L, M 1 D, the right eye being used for distant vision and the left eye for near vision. The refraction was corrected and both eyes brought into use for all purposes. Cocaine was dropped into the eyes at regular intervals and bromide of potassium administered internally. She at once began to improve and within a fortnight the spasm was scarcely noticeable.

Case 21. *Corneal Ulcer.*—A cook, æt. 21, was admitted into the Bath Eye Infirmary on April 29, 1890 with a sloughing ulcer of the cornea, which dated from an attack of influenza in March. Its onset, she said, was sudden one night, the pain waking her up. The ulcer was treated with boracic massage and healed up in about three weeks.

CASE 22. *Glaucoma.*—A weaver, æt. 74, was attacked by influenza in February, and before he got over it the R eye became very painful and inflamed. He neglected it till May 20th, when the condition was T + 1, mydriasis, no vision, details of fundus invisible. An iridectomy was performed and the pain, tension, and inflammation gradually subsided. He was discharged June 5th.

In these cases there seems to be something more than mere coincidence in the relationship of the eye affection to the attack of influenza. Troubles of the accommodation are so frequent a sequel of other debilitating diseases—such as typhoid, relapsing fever, etc., that it is scarcely a matter of surprise to find influenza followed by ophthalmic complaints. Corneal ulcers, keratitis, iritis, etc., are possibly instances of what Mules calls tropho-neuroses. In hypermetropia the ciliary muscle, which in health may have been sufficient, by an effort of accommodation which was inappreciable, to bear the ordinary daily strain, can, after an exhausting disease, such as influenza, no longer fulfil its duties. The accommodation, which had hitherto been capable of neutralizing the error of refraction, is now unequal to the task, and the tendency is for the Ht. to be converted into Hm. As a consequence the usual asthenopic and accommodative symptoms common to the error are manifested, and the equilibrium can only be re-established by resorting to correcting lenses.

But though errors of refraction, plus post-influenza debility, may result in accommodative troubles, yet it is evident that ocular disease cannot be accounted for in this way in those cases in which there is no error of refraction, nor in those cases in which the resulting disease is not one that has any connection with the refractive or accommodative apparatus. It must, however, be difficult to say in many cases what the true relationship really is. Take, for instance, cases

11 and 22 in the above series, what are the successive pathological steps which, beginning in influenza, terminate in glaucoma? We know that influenza leaves troops of sequelæ, or rather we know that it is usual to account for many minor illnesses that have been preceded influenza by fathering them upon the epidemic. We know too that great physical and mental depression characterize influenza, and we know also that glaucoma frequently follows depression whether of body or mind, but the exact process is too subtle for analysis, and it is less easy to *account for* than it is to *know.*

A CASE OF INTERNAL STRABISMUS WITH CROSSED DIPLOPIA.

By A. B. OSBORNE, M.D.,

OPTHALMIC AND AURAL SURGEON, CITY HOSPITAL, HAMILTON, ONT.

Early in the present year J. V., æt. 21, came to me on account of a slight internal strabismus. Two years previously a competent oculist had divided the internal rectus of each eye, resulting in a moderate improvement.

The vision in the left eye was $\frac{6}{6}$, right $= \frac{6}{9}$. He could fix with either eye indifferently, although he most frequently used the left. Under atropine R V $= \frac{6}{36}$; $+ 1.5$ D $= \frac{6}{6}$ partly, L V $= \frac{6}{18}$; $+ 1.5$ D $= \frac{6}{6}$, fundi normal: this correction was ordered. After wearing the glasses for three weeks, with the eyes under atropine—no diminution of the squint having taken place—I divided the right internal rectus, leaving the eyes apparently straight. He now complained of diplopia, which proved upon examination to be crossed, any attempt to fuse the double images causing the strabismus to return; and when he reported the images fused the squint was as great as before the operation. By the cover test there was a slight but distinct movement outward, in order to fix, showing that there was still a convergent squint, notwithstanding the crossed diplopia. The strabismus now gradually returned to about the condition in which I first saw it, with the exception that he could produce crossed diplopia at will. A few days after the operation, I noticed for the first time a difference in the horizontal planes of the eyes, notwithstanding which he reported the double images on a level.

The strabismus having now resumed its original position, I undertook what proved to be a most arduous task, viz., to teach him to have homonymous diplopia; at the end of a week's practice he was able to produce occasional homonymous images, but could not hold them long, as they became crossed. The closest attention to his eyes revealed no movement at the time when he declared the diplopia to change from homonymous to crossed, although the images, according to his statement, must have moved a distance of nearly six feet. With crossed diplopia the images were on a level and steady ; with homonymous diplopia the right was much the higher and unsteady. Two weeks' practice resulted in almost constant homonymous diplopia, an 8° prism bringing the images about level ; but this could not be measured accurately as the images became crossed when brought near the same level.

One month after the first operation I divided the left superior rectus. For a few days he had little control over the images, the diplopia becoming crossed in spite of him ; but he noticed that the homonymous images were about on the same level, while there was nearly the same difference between the height of the crossed images as had previously existed between the homonymous. A few days later, the eyes having become settled, he had continuous homonymous diplopia, the right image being still slightly higher than the left, 1° prism bringing them even.

I now began to practise approximating the homonymous images with prisms, and encountered great difficulty, even a weak prism at first causing the images to cross. After a month's practice he thought a pair of 5° prisms about fused the images, but that belonging to the right eye was unsteady and floated about. While using these prisms he became aware of a third image, which came into view when the homonymous images were brought close together. This third image came into view when he fixed with either eye and was always crossed, belonging to the deviating eye, viz., when fixing with the left the third image was below and on the left, when fixing with the right the third image was above and on the right, thus corresponding to the original crossed images, their plane having been altered by the division of the superior rectus. The triplopia was constant while wearing a pair of 5° prisms.

Having now accustomed himself to the proximity of the homonymous images, and being able to hold them without crossing, I divided the left internal rectus. After this operation the images were so close that owing to the unsteadiness of the right they occasionally merged into one. It is noteworthy that although the tendency to fusion of the crossed images had been strong enough to cause a recurrence of the squint after the first operation, yet, notwithstanding the proximity of the correct images and my constant injunctions to endeavour to fuse them, he not only has no tendency to fusion, but is unable to maintain single vision when they become fused of their own accord.

The present condition is almost similar to that just after the last tenotomy ; the images partly overlap each other, the right is still rather unsteady and by alternately covering each eye there is a very slight motion of adjustment. He is learning to ignore the third image. There is no tendency to fusion.

It is worthy of remark, that the functions of the macula were assumed by eccentric portions of each retina, and not by that of the deviating eye alone.

In many respects this case is a corroboration of Stevens' remarks upon strabismus in the December number (1889) of these ARCHIVES.

A REPORT OF TWENTY-FIVE CATARACT EXTRACTIONS, WITH REMARKS.

By JAMES L. MINOR, M.D., Memphis, Tenn.

I HAVE collected the notes of twenty-five consecutive cases of cataract operated upon by me during the past few years. The first eleven cases were treated at the New York Eye and Ear Infirmary during my connection with that institution[1]; the remainder—fourteen cases—were treated in Memphis. Of the 25 cases operated upon, 13 men and 5 women were white; 3 males and 4 females were colored.

The oldest was 82 years; the youngest, 41; the average, 63. The results were uninfluenced by age.

The general health was good or fair in all.

The cataract was mature in 22 cases, and hypermature in 3. Equally good results were obtained in the two conditions.

The right eye was operated upon in 12 cases, and the left in 13.

The greatest duration of cataract was 23 years; the shortest, 6 months; the average, 2 years.

All eyes were flushed with a saturated solution of boric acid before the operation. No other antiseptic precautions were practised at this time further than scrupulous cleanliness.

Preliminary iridectomy, with trituration of the lens, was practised in 4 cases—good results being obtained.

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[1] The reports of these cases were copied from the records of the New York Eye and Ear Infirmary, for me, by the then House-Surgeon, Dr. F. N. Lewis.

TABULATED REPORT

Name, Nativity, and Residence.	Age.	General Health.	Corneal Diameter.	Size and Dilatability of Pupil.	Quality and Duration of Cataract.	Functional Examination.	Date of Operation.	Operation. Method and Incidents. Anæsthetics.
Oliver Dexter, Jersey City. Case I.	69	Good, except emaciated.	Normal.	Normal.	O. S. 2 years.	Normal.	Aug. 8, 1881.	Ether. Graefe's mod. lin. section; medium hemorrhage into anter. chamber. Iridectomy large. Cystotomy vertical, pressure required to cause lens to escape. Some cortical remaining which was removed. Some blood left in anter. chamber.
John Welsh. Case II.	60	Fair.	N.	N.	Senile. O. D.	N.	June 22, 1883.	No ether. Graefe's mod. linear upwards. Section large. Free iridectomy. Peripheral capsulot. Lens dislocated down and backwards. Delivery with spoon. Slight escape of vitreous.
Amanda Jackson. (col.) Case III.	60	Fair. Syphilitic.	N.	N.	Mature. O. S.	N.	July 13, 1883.	Ether. Graefe's mod. linear upwards. Small incision. Medium iridectomy. Good deal cortical. Extrusion easy. Vitreous presented in wound, did not escape. No accident.
Henry Lutja. Case IV.	72	Poor. Phthisical.	N.	N.	Mature. O. D.	N.	July 16, 1884.	Chlor. and ether. Graefe's mod. linear upwards. Section small. Preliminary iridect. (Förster), March 28, 1884. Peripheral cystotomy. Abundant soft cortical. Delivery easy. Some cortical left.
Catharine Hamstahl. Case V.	49	Good.	N.	N.	Mature. O. D.	N.	Oct. 13, 1884.	Cocaine 2 %. Graefe's mod. lin. upwards. Section large. Förster, Sept. 1, 1884. T. Cystotomy. Delivery easy; much cortical remaining; no pain. Boric acid washing; anæsthesia perfect.
Wm. McCormick. Case VI.	70	Good.	N.	N.	Mature. O. S.	N.	Nov. 21, 1884.	Cocaine 4 %. Peripheral incision upwards. Section medium. Small iridectomy. Slight hemorrhage from iris. Peripheral capsulotomy. Delivery slow. No accident.
Charles O'Blimis. Case VII.	58	Good.	Normal.	Normal.	Mature. O. D.	Normal.	Dec. 5, 1884.	Cocaine. Peripheral section upwards. Corneal wound large. Förster, Nov. 10, 1884. Peripheral cystotomy. Delivery easy. Pupil clear. No accident. Perfect anæsthesia.
Thomas H. Evans. Case VIII.	82	Good.	N.	N.	Mature. O. S.	N.	Dec. 5, 1884.	Cocaine 4 %. Peripheral section upwards. Corneal incision large. Broad iridectomy. Hemorrhage into anterior chamber. Peripheral cystotomy. Delivery easy. Complete collapse of cornea. Perfect anæsthesia.

OF THE CASES.

Course of Healing Process and Treatment.	Length of Treatment.	Vision at time of Discharge. Far and Near.	Secondary Operation. Date, Kind, Course, and Result.	Ultimate Vision, with Date of Testing.	Remarks.
Aug. 11th.—Patient last night had delusion that she was being smothered to death from escape of gas into room; became quite frantic, tearing bandage off and screaming at top of her voice. She was given dose K. Br. and whiskey ʒi, which soon quieted her. Aug. 13th.—The same thing last night, and patient quite unmanageable. Iritis set in; treated with atropine. Allowed to get up and see company, and insanity disappeared. Aug. 19th.—Eye quiet, posterior synechia at lower part.	11 days.	Aug. 19. 1881. $V = \frac{6}{200}$, with $+ 8$ D's $= \frac{10}{100}$. Sept. 28, 1881. $V = \frac{6}{200} + 12$ D's $= \frac{20}{60}$.	March 24, 1882. With $+ 4$ in. $\frac{20}{50}$. With $+ 2\frac{1}{2}$ reads Sn. D. = 1 at 10 in.	$\frac{20}{40}$	
Normal. Left without permission and before vision could be taken.		$V = \frac{20}{\text{XL}}$ probably, as there was no membrane and perfectly clear pupil.			
Every thing went well until 5th day, when iritis developed on 7th day of a purulent nature. Put on hot applications and atrop. This was continued until 15th day, when process slowly subsided, leaving pupil filled with dense membrane.	18 days.	V = perception of light. Dense membrane in pupil.			
Normal.	12 days.	$V = \frac{20}{\text{LXX}}$			
Normal.	17 days.	$V = \frac{20}{\text{LXX}}$			
Normal.	16 days.	$V = \frac{20}{\text{LXX}}$ $+ 3\frac{3}{4}$			
Normal.	12 days.	$V = \frac{20}{\text{XL.}} + 10$ D's.		$V = \frac{20}{\text{XX.}} + 10$ D's $\supset + 1.50$ c. Ax. 120°. Reading $+ 15$ D. $\supset + 1.50$ D.c. at 120°.	
Wound opened on 5th day. Bandage atrop. once daily. Wound closed on 15th day. Otherwise process of healing normal.	26 days.	V = counts fingers. Dense pupillary membrane obscuring sight.	Needled Jan. $V = \frac{20}{\text{C}}$ with $+ 10$ D. Reading $+ 16$ D. Jan. 21, 1885.		

Name, Nativity, and Residence.	Age.	General Health.	Corneal Diameter.	Size and Dilatability of Pupil.	Quality and Duration of Cataract.	Functional Examination.	Date of Operation.	Operation. Method and Incidents. Anæsthetics.
Nathaniel Parks. Case IX.	57	Good.	N.	N.	Mature. O. D.	N.	Dec. 12, 1884.	Cocaine 4 %. Peripheral section upwards. Förster, Nov. 7, 1884. Peripheral cystotomy. Delivery easy. No accident. A. B. wash.
Mary Schlarf. Case X.	61	Good.	N.	N.	Mature. O. D.	N.	April 10, 1885.	Cocaine 4 %. Peripheral section upwards, with conj. flap. Medium iridectomy. Peripheral capsulotomy. Extrusion easy. Counted fingers. No accident.
Charles O'Blimis. Case XI.	58	Fair.	N.	N.	Mature. O. S.	N.	May 1, 1885.	Cocaine 4 %. Peripheral section upwards. Preliminary iridectomy (Förster). Peripheral capsulotomy. Extrusion easy and without accident. Counted fingers.
Mr. J. P., U. S. Male. White. Case XII.	41	Fair.	N.	N.	Hard cataract right eye. 4 years.	N.	June 29, 1886.	Peripheral section upwards. Good iridectomy. Peripheral cystotomy, and lens dislocated downwards. Vitreous presented and lens delivered with spoon. Slight loss of vitreous. Cocaine.
Capt. H., U. S. White. Case XIII.	70	Good.	Normal.	Normal.	2 years. Hard cataract. Hypermature.	Normal.	Oct. 5, 1886.	Right eye. Graefe's modified peripheric linear section upward, with apex well in cornea. Good iridectomy. Peripheric cystotomy. Slight loss of vitreous, and lens delivered with spoon. Cocaine.
Mrs. R., Ireland. White. Case XIV.	65	Good.	N.	Large but otherwise normal.	6 months. Hard cataract.	N.	Feb. 13, 1887.	Right eye. Graefe's modified linear section, upward. Good iridectomy. T-shaped cystotomy. Lens large, and following its delivery was loss of vitreous. Cocaine.
S. M., U. S. Female. Colored. Case XV.	60+	Good.	N.	N.	1 year. Hard cataract.	N.	March 12, 1887.	Left eye. Graefe's modified linear section upward. Large iridectomy. Peripheric cystotomy. Delivery of lens easy and without accident. Cocaine.
B. H., U. S. Negro. Man. Case XVI.	60+	Good.	N.	N.	2 years. Hard cataract.	N.	Oct. 7, 1887.	Right eye. Operation as above. Cocaine.
H. J. T., U. S. Male. White. Case XVII.	56	Good.	N.	N.	2 years. Hard cataract.	N.	Jan. 20, 1888.	Left eye. Operation as above. Cocaine.
M. O. P., U. S. Male. White. Case XVIII.	60	Fair.	N.	N.	1 year. Hard cataract.	N.	Feb. 12, 1888.	Left eye. Preliminary iridectomy with trituration of lens one month before. Graefe's modified linear section upward. T-shaped cystotomy. Lens delivered with ease. Cocaine.
M. M., U. S. Female. Negro. Case XIX.	70	Fair.		Normal.	2 years. Hard cataract, hypermature.	Normal.	Nov. 5, 1888.	Left eye. Graefe's modified linear section upward. Large iridectomy. Peripheric cystotomy. Delivery of lens easy. Cocaine.
S. R., U. S. Male. Negro. Case XX.	65	Good.		N.	1 year. Hard cataract.	N.	Dec. 10, 1888.	As above. Right eye. Cocaine.
C. B., U. S. White. Male. Case XXI.	67	Fair.		N.	4 years. Mature cataract.	N.	Jan. 6, 1889.	Left eye. As above. T-shaped cystotomy. Delivery easy. Cocaine.

Course of Healing Process and Treatment.	Length of Treatment.	Vision at Time of Discharge. Far and Near.	Secondary Operation. Date, Kind, Course, and Result.	Ultimate Vision with Date of Testing.	Remarks.
Normal.		$V = \frac{20}{xxx.} + 10$ D. ⊃ + 1.50 D.c. ax. 125°.			
Normal.	14 days.	$V = \frac{20}{cc.}$ with + 3½. Some cortical matter + capsule in pupil.			
Normal.	38 days.	$V = \frac{20}{c} + 3\frac{3}{4}$			
Normal until 8th day, when sharp attack of iritis occurred, which yielded to treatment.	3 wks.		Membrane divided with cutting needles 2 months after extraction.	Feb. 21, 1887. $\frac{20}{50}$ with + 14^{oo} D. For reading + 18^{oo} D.	
Normal. Eye kept bandaged until 4th day, when lids were kept closed with adhesive plaster.	10 days.	$\frac{20}{200}$	May 30, 1886. Secondary membrane divided with sickle knife. Result good. $\frac{20}{200}$.	Nov. 25, 1887. $\frac{20}{70}$, with + 10 D. With + 14^{oo} D, reads No. IV. Sn.	
Normal.	1 week.			March 18, 1887. $\frac{20}{70}$ with + 12^{oo} D. With + 15^{oo} D. sees to thread needle.	
Normal.	1 week.	$\frac{10}{200}$	April 20, 1887. Membrane divided with knife-needle. Good result.	May 10, 1887. $\frac{20}{100}$ with + 11 D. Small type with + 15 D.	To report later for another division of membrane.
Normal, except slight iritis on 4th day, which yielded to treatment.	2 weeks			$\frac{20}{50}$ with + 10 D. Reads with 14 D.	
Normal.	10 days.			March 12, 1888. $\frac{20}{30}$ with + 9 D. S. ⊃ + 1.75 D. C. at 180°. Reads No. II J with + 14 D. S. ⊃ + 1.75 D. C. at 180°.	
Normal.	10 days.			May 10, 1888. $\frac{20}{30}$ + with + 10 D. S. ⊃ + 1.50 D. C. at 125°.	
Normal. Slight iritis on 5th day, which yielded to atropia and warm-water bathing.	2 wks.		Membrane divided with cutting-needles about two months after extraction.	Dec. 10, 1888. $\frac{20}{100}$ with + 12 D. For reading, + 15 D.	
Normal.	10 days.			Jan. 15, 1889. $\frac{20}{200}$ with + 11 D. For reading + 15 D.	To report later for secondary operation.
Normal.	1 week.			Feb. 27, 1889. $\frac{20}{50}$ + 10 D. For reading + 14 D.	

Name, Nativity, and Residence.	Age.	General Health.	Corneal Diameter.	Size and Dilatability of Pupil.	Quality and Duration of Cataract.	Functional Examination.	Date of Operation.	Operation. Method and Incidents. Anæsthetics.
J. F., U. S. Male. Colored. Case XXII.	60	Good.	N.	N.	23 years.	N.	March 7, 1889.	Right eye. As above, with peripheric cystotomy. Cocaine.
Mrs. A. C. B., U.S. White. Case XXIII.	77	Fair.	N.	N.	4 years. Mature cataract.	N.	April 9, 1889.	Left eye. Operation as above. Cocaine.
Mrs. O'B., U. S. White. Case XXIV.	70	Good.	N.	N.	1 year. Mature.	N.	Oct. 10, 1889.	Left eye. As above. Cocaine.
J. R., U. S. Colored. Female. Case XXV.	62	Good.	N.	N.	3 years. Hypermature.	N.	April 4, 1890.	As above. Left eye. Cocaine.

Course of Healing Process and Treatment.	Length of Treatment.	Vision at Time of Discharge. Far and Near.	Secondary Operation. Date, Kind, Course, and Result.	Ultimate Vision, with Date of Testing.	Remarks.
Normal.	2 wks.			May 8, 1889. $\frac{20}{30}$ with + 11 D. For near work + 13 D.	
Normal. Slight iritis about 5th day. Eyes closed with adhesive plaster after 5th day.	10 days.			May 28, 1889. $\frac{20}{70}$ with + 7 D. For near work + 10.	
Normal.	1 week.		Needled Nov. 16th.	Dec. 13, 1889. $\frac{20}{40}$ with + 11 D, ⌢ + 1 D.c. at 140°. For reading + 14 D with above cylinder.	
Normal.	10 days.			April 28, 1890. + 12^{00} D. S, V = $\frac{20}{20}$? + 15^{00} S. Reads I Sn. at 10".	

With that exception, the operation known as Graefe's modified linear extraction was employed in all cases.

A good free iridectomy was obtained in every instance.

Peripheral cystotomy was performed in 18 cases; T-shaped in 6, and vertical in 1.

The lens was dislocated during the cystotomy in 2 cases, with slight loss of vitreous, and delivery was effected with a spoon in each instance, giving good results.

Vitreous escaped in 2 other cases, in one of which the lens was removed with a spoon, and good results obtained.

One case was operated upon without an anæsthetic; ether was employed in 2, chloroform and ether in 1, and cocaine in 21.[1]

The compress bandage was employed in all cases, and, in most instances, it was continued as long as it was desired to keep the lids closed, or about a week, when a shade was substituted. In a few cases where the bandage was uncomfortable it was removed altogether, and the lids closed with isinglass plaster and the eyes protected with a wire-cloth shade.

It has always been my habit to dust pulverized boric acid freely over the pad which lies next the eye and underneath the cotton compress, when applying the compress bandage. The moisture which escapes from between the lids dissolves the acid as it does so, and in that way an aseptic condition is kept up.

The healing process was normal in 17 cases.

Iritis, about the 5th day, occurred in 7 cases. This yielded readily to treatment in all but 1, in which a dense pupillary membrane formed.

The wound opened on the 5th day in 1 case, but healed and gave a good result under proper treatment.

A mild form of insanity appeared on the 3d day in 1 case, which was probably dependent upon the new surroundings, the confinement, the maintenance of the recumbent posture, and the bandage, for it all disappeared when these conditions were rectified.

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[1] It may be interesting to know that case No. 5 in this exhibit was the first cataract extraction performed in the United States under the influence of the, then, new anæsthetic, muriate of cocaine. See *New York Medical Record*, October 18, 1884.

The average duration of treatment was 14 days.

A secondary operation was performed in 7 cases—6 having had peripheric, and 1 vertical, cystotomy.

Four cases were to return for a secondary operation, but failed to report.

Twenty-four cases had useful vision restored, and a large percentage of these saw almost as well as they had ever done.

One case only was not benefited by the operation, for a dense membrane occupied the pupil and coloboma. Useful vision could probably have been restored by a secondary operation, but the patient did not return for it.

3 cases had vision of $\frac{20}{20}$
3 " " " " $\frac{20}{30}$
2 " " " " $\frac{20}{40}$
4 " " " " $\frac{20}{50}$
6 " " " " $\frac{20}{70}$
4 " " " " $\frac{20}{100}$
2 " " " " $\frac{20}{200}$
1 " " " " P. L.

A CASE OF HEMORRHAGE INTO THE VITREOUS AFTER A CATARACT EXTRACTION.[1]

BY H. KNAPP.

MR. PRESIDENT: When at the last meeting of this society, in the discussion on hemorrhage into the vitreous after cataract extraction—so judiciously introduced by yourself—you asked me my experience on this subject, my answer was: "I have never seen a case, either in my own practice or in that of anybody. I consider this only as a lucky chance, for, according to the usual course of events, I may have two cases the next month." What I had apprehended has, in part at least, but too soon occurred.

On November 1, 1890, I removed a Morgagnian cataract from the left eye of Mrs. Ann Corcoran, a seventy-year-old woman of this city. She perceived the light of a candle all around the eye, but was not able to localize it anywhere, a condition not unusual in milky cataract, you know, and not always connected with serious changes in the deeper structures of the globe. The eyeball, even after thorough cocainization, was hard, and I said that I probably would have to combine the extraction with an iridectomy. After a normal upper section, I opened the capsule under the upper portion of the iris, whereupon the anterior chamber was flooded with a milky fluid. I expelled the very small brown nucleus without difficulty, gave the eye a few minutes' rest, and then cleansed the anterior chamber by gentle rubbing with the lower lid. The iris fell back, the pupil was central and round, and the patient counted fingers correctly and readily. I instilled eserine, but when I re-inspected the wound, about a minute later, in order to bandage the eye, I found that by the least motion of the lid the iris would push toward the wound. I therefore made a small iridectomy, which was done without difficulty and without bleeding. I should mention that no bleeding occurred during the whole operation. In adjusting the corners of the coloboma, I noticed that a bead of vitreous had a tendency to present in the centre of the wound. Under gentle pressure with a pad of absorbent cotton the eye was kept closed for a few minutes, but when opened again a broader bead of vitreous crowded into the section, yet without protruding. I said to the by-standers this looked as if intraocular hemorrhage would occur. A regular compressive bandage was applied, and the patient put to bed. She complained of considerable pain in her eye. I told her this was not unusual, and she would soon feel easier. So she did, and I went into another room to perform some other operations. In about half an hour the nurse reported that the old woman felt more pain, had vomited several times, and blood flowed through the bandage down her temple. I removed the dressing, which was soaked with blood, and found a large clot of blood, with here and there some viscid soft liquid, protruding through the palpebral fissure. On gently raising the upper lid, I saw that the clot was lying in the widely gaping corneal section. I removed it as well as I could, dropped a solution of bichloride of mercury, 1–10,000, into the conjunctival sac, washed the outside of the lids carefully with the same solution, and applied an antiseptic dressing (sublimate gauze and absorbent cotton moistened with a 1–5,000 solution of corrosive sublimate), held in position by an elastic flannel roller. The pain soon subsided, and there was no further hemorrhage.

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[1] Read before the Section of Ophthalmology at the New York Academy of Medicine, November 17, 1890.

The next day, on changing the dressing, which was still soaked with blood but dry, a clot lay in the opened section. It was carefully removed with a pair of forceps. There was no swelling of lid or conjunctiva, and the cornea was clear ; the anterior chamber was full of blood. After a solution of corrosive sublimate had been instilled, the same dressing was applied as the day before.

The dreaded suppuration did not occur. The patient felt comfortable, the blood-clot in the wound contracted, the section closed, the cornea remained transparent, and the blood in the anterior chamber gradually absorbed.

At the end of two weeks after the operation the patient desired to be transferred to some other hospital. The wound being substantially healed, she was discharged with a blind but painless and non-disfigured eye.

The microscopic examination showed the clot which had been hanging out of the corneal section to consist of a mass of blood corpuscles, in which vitreous, lens capsule, and pieces of retina, especially of the pars ciliaris, were embedded.

What caused the retrohyaloid hemorrhage in this case after a perfectly smooth extraction I am at a loss to surmise. As to its course and termination, this case has gone against the rule, which is suppuration and panophthalmitis. This unfortunate issue has been noticed with such frequency as to induce some operators to advise immediate enucleation when hemorrhage into the vitreous sets in. In the above-described case the hemorrhage was as copious as it could be, and evidently originated in the posterior segment of the eye, for it expelled vitreous, retina, and lens capsule. Nevertheless, the recovery was by primary union. As, for practical purposes, we may confidently consider all suppuration caused by the action of pyogenic germs, suppuration will not occur in any wound as long as we can keep pyogenic germs out. Consequently an extraction wound, even if complicated with severe intraocular hemorrhage, is not necessarily doomed to suppurate. I would therefore treat such cases exactly as the one detailed above: remove the blood carefully, wash the conjunctival sac out with a mild antiseptic, sterilize the outside of the lids in like manner, and apply an antiseptic dressing. Change the dressing once or twice daily, according to the presence of discomfort and more or less discharge. If the beginning of panophthalmitis manifests itself, enucleate without delay, otherwise spare the eye.

CASE OF HERNIA OF IRIS OCCURRING ON THIRD DAY AFTER CATARACT EXTRACTION WITHOUT IRIDECTOMY; HERNIA REPLACED; RECOVERY.

By HERBERT HARLAN, A.M., M.D., Baltimore.

THE following case bearing on the time of complete healing of a corneal wound, and on the liability to prolapse of the iris, is perhaps of sufficient interest to justify being put on record.

A somewhat similar case resulting from an injury on the sixth day is reported by Dr. Knapp in THESE ARCHIVES Vol. XVII., page 55 (case 67).

M. Y., æt. fifty-eight, having a mature cataract in the left eye and some clouding of the lens in the right, was operated under cocaine anæsthesia July 16th; the left lens being extracted through large upward peripheral incision without iridectomy. Operation smooth. A solution of eserine was instilled, and the left eye closed with strip of isinglass plaster, the right being left open. Eserine again instilled at bedtime. There was absolutely no reaction, patient being up and about the room daily. At my visit on the 19th the strip had become so loosened that it was removed. There was no redness, and the line of corneal incision seemed perfectly healed. The pupil was round and central. No drops had been used after evening of first day. Patient was seemingly doing so well that both eyes were left open. At my visit on the 20th I was surprised to find that the corneal wound had opened for a space of about one line, and that there was a prolapse of the iris. As the hernia was of so recent occurrence, I determined to endeavor to replace it. Having let out the aqueous with a narrow cataract knife, this was readily done with the aid of a small tortoise-shell spatula, when the pupil again became round. Eserine was continued three times a day for two more days, and patient left the hospital for his home in an adjoining county at the end of two weeks with central pupil and good vision, $\frac{20}{C}$. He has not yet returned, as promised, for final adjustment of glasses, probably because as yet the sight is still good in the right eye.

The points of special interest are:

First, that the corneal wound is not safely healed at the end of three days.

Secondly, that a recent hernia may be safely replaced instead of being clipped off as is usually done; and

Thirdly, that while it is not advisable to do so, yet a great many more liberties may be taken with eyes after cataract extraction than many of us have been taught to believe.

A CASE OF BILATERAL HEMIOPIA.

By Prof. C. SCHWEIGGER, Berlin.

Translated by Dr. Ward A. Holden, New York.

BILATERAL hemiopia is so rare that the reporting of even single observations seems called for, and I am the more persuaded to record this case, since it resembles Förster's case[1] in some particulars and in others differs from it.

H. von Sch., æt. seventy-five, had been under my treatment for several years for conjunctival catarrh and nuclear cataract of the right eye (L : Em., $V = \frac{9}{12}$; R : — 1.25, $V = \frac{6}{36}$), when in September, 1888, a hemiopic defect appeared suddenly in both left halves of the visual fields, without the occurrence of other disturbances, such as loss of consciousness, hemiparesis, etc. The central vision remained unchanged, and the ophthalmoscopic appearance was normal, as is the rule in these cases. In August, 1889, the right halves of the visual fields failed suddenly, as the left halves had failed before. The patient supposed himself to be quite blind, and his movements also gave this impression, yet, as in Förster's case, a small central field of vision was preserved. In the street he found that he still saw the dials on the clock-towers, and other distant objects, as, for example, single words in the shop-signs. A careful examination showed that at a distance of 3 or 4 metres the patient could just recognize the dial of a clock on the wall and distinguish the figures on it. Corresponding to the size of the dial (20 *cm*) and the figures, there remained a visual field of 0° 22′ at the most, in which his former acuteness of central vision was preserved. This vision was naturally useless for the perception of near objects, since a field of 22′ is much too small for this purpose. It should be stated further that the patient had not lost the perception of light completely in the region of the hemiopic defect, since movement of the hand was perceived excentrically.

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[1] *v. Græfe's Arch.*, xxxvi., 1, p. 94.

The chief point which this case presented in common with that of Förster, was the preservation of a limited central field of vision, although this was much smaller in my case than in his. This fact proves the preservation of the central acuteness of vision and the well-known inflection found in the defect in the visual field near the point of fixation, to be due to a particular arrangement in the central apparatus. The points of difference were the following:

1. In Förster's case the loss of the second halves of the fields of vision occurred gradually in the course of two days, while in my case it came suddenly.

2. The weakness of memory and the loss of the sense of locality, which Förster observed, were not found in my case. When the patient came to me, blind, except in a minimal field, he appeared familiar with the localities which he had previously known. Apart from the further development of the cataract, no change has taken place up to this time, October, 1890.

A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF PANOPHTHALMITIS.

By Prof. JOS. SCHÖBL, Prague.

Translated by Dr. Ward A. Holden, New York.

(*With Plates X.-XV. of Vol. XXI., German Edition.*)

HAVING selected from my collection twenty-seven eyes where the clinical diagnosis of panophthalmitis from purulent choroiditis had been made, and the enucleations had been performed in various stages of the disease, I subjected them to a careful examination.

The eyes were all cut in a sagittal direction and stained for the most part with hæmatoxylin. In most cases a careful injection of the blood-vessels was made according to my method immediately after the enucleation, before the ball was put into the hardening fluid, and only a few times was this unsuccessful. Special staining for bacteria was omitted as being unreliable.

I shall first describe in brief these cases, arranging them according to the etiological factor which caused the panophthalmitis, and then draw the conclusions which naturally follow as to the pathological anatomy of panophthalmitis.

Panophthalmitis Following Various Corneal Injuries.

a.—VULNUS PERFORANS CORNEÆ CENTRALE.

Case 1 (Fig. 1).—Z., æt. fifty-six, penetrating wound through the centre of the cornea and the lens, without injury to the iris, caused by a piece of unclean iron. Progressive inflammation. Extirpation of the swollen and protruding eye in the stage of fully developed panophthalmitis. Pus escaped through the perforated cornea.

After the successful extirpation the eye was hardened in equal parts of Müller's fluid and saturated solution of picric acid, then washed in distilled water and later in dilute alcohol, then in stronger and finally absolute alcohol, then brought in oil of cloves under the air-pump, finally imbedded in celloidin and cut in sagittal sections.

Anatomical Examination.—The eye is enlarged and pear-shaped, the sagittal diameter being 32 *mm*, the equatorial 24 *mm*. It is filled with masses of pus, no lens or vitreous can be discovered, and only a small bit of the posterior portion of the retina near the papilla remains (Fig. 1). The choroid in its whole extent is but slightly changed. The retinal pigment epithelium is preserved from the papilla to the ora serrata. Lying on the pigment layer is a layer of granulation tissue uniformly supplied with vessels which pass as short branches from the stratum vasculosum of the choroid and perforate the epithelium. This granulation tissue consists of leucocytes, scanty intervening substance, new-formed vessels, and traces of connective tissue, and is identical with the hyperplastic choroiditis described by me in vol. xix. of these ARCHIVES.

The stroma of the choroid proper is uniformly infiltrated with leucocytes. The granulation tissue is thickest on the pars plicata of the ciliary body. The iris, which is adherent to the cornea, is also covered with a thick layer of granulation tissue, having a number of new-formed vessels from the vessels of the iris.

The sclera in its outer layers contains leucocytes closely packed, and many new-formed vessels. The same exuberant granulation tissue passes in tongue-like masses into the outer layers of the sclera.

The anatomical diagnosis is therefore: retinitis purulenta; irido-cyclo-choroiditis (uveitis) hyperplastica; tenonitis hyperplastica; conjunctivitis hyperplastica.

A single glance at a preparation of this eye or at the drawing, Fig. 1, is sufficient to show that it was primarily and chiefly a retinitis purulenta, which ran its course with the clinical symptoms of a violent acute traumatic panophthalmitis, and that the uveal tract became affected later and less severely, and was in no way the occasion of the severe clinical symptoms of panophthalmitis which were observed.

Case 2.—E. S., æt. fourteen, centre of the cornea pierced by a small arrow, beginning cataract, yellow reflex from the fundus. Gradually there developed the characteristic picture of acute panophthalmitis with enlargement of the ball, protrusion, immobility, chemosis of the conjunctiva and lids, complete blindness, and severe pain. Extirpation two weeks after the injury.

The examination revealed conditions almost identical with those of the previous case: retinitis purulenta; irido-cyclo-choroiditis (uveitis) hyperplastica; scleritis, tenonitis; conjunctivitis hyperplastica.

In this case also there can be no doubt but that it was originally an acute traumatic retinitis which ran its course under the clinical picture of a panophthalmitis, and that the slow inflammatory changes in the choroid were of later date and lesser importance.

b.—CORPORA PEREGRINA PER CORNEAM IN BULBUM PENETRANTIA.

I have before me successful preparations of three eyes in which panophthalmitis occurred after the entrance of foreign bodies through the cornea. In all three cases fragments of gun-caps passed through the centre of the cornea and the lens into the interior of the eye, without wounding the uveal tract. These cases have been previously described in the *Centralbl. f. prakt. Augenheilk.*, March, 1889, in a paper on primary purulent retinitis.

Case 3.—S., æt. eight, piece of gun-cap passed through cornea and lens into the vitreous. On the third day panophthalmitis incipiens, eye enucleated on the fourth day.

Microscopic Examination.—Choroid normal, retina showing signs of severe inflammation, the nerve-fibre layer greatly thickened, its vessels dilated and distended with blood, and the whole retina infiltrated with leucocytes. The hyaloid is detached from the retina and an intervening space is formed, which is filled with a thick layer of pus and masses of coagulated fibrin. The lens is displaced forward and infiltrated with leucocytes. The cornea, in the region near the wound, the iris, and the vitreous, are also infiltrated with leucocytes.

The anatomical diagnosis is : retinitis purulenta acuta traumatica ; hyalitis traumatica ; phakeitis (infiltratio lentis) traumatica ; irido-keratitis traumatica. No signs of choroiditis.

Case 4.—P., æt. six, fragment of gun-cap passing into the right eye through the centre of the cornea. The clinical picture and the diagnosis were similar to those of the foregoing case.

Enucleation ten days after the injury. Microscopic examination showed a dense mass of pus in the vitreous chamber, in which here and there single bits of the retina could still be distinguished.

The choroid was almost normal, but moderately thickened and slightly infiltrated with leucocytes.

In this case also there could be no doubt but that the primary and principal affection had been an acute traumatic retinitis showing the signs of beginning panophthalmitis.

Case 5.—K., æt. ten, left eye wounded in a similar manner, and a preventive enucleation made twelve days later.

The anatomical condition resembled that of Case 3, except that the anterior chamber was preserved.

Wound in cornea cicatrized. Traumatic cataract. Retina in toto greatly thickened and closely infiltrated with leucocytes, and covered on its internal surface by a thick layer of pus.

Here also the choroid was almost intact, and the disease has been undoubtedly a purulent retinitis with the symptoms of beginning panophthalmitis.

c.—COMBUSTIO CORNEÆ CUM FERRO LIQUEFACTO.

Case 6 (Fig. 2).—J., æt. twenty-seven, injured by melted iron spurting into his right eye. The iron was removed in the form of a thin plate, which had formed an accurate mould of the cornea and limbus.

Seventeen days later the patient was admitted to the hospital. The eye had been red and painful, lids swollen, V = 0. At this time the eye was enlarged, prominent, and immobile. The cornea was grayish-white and vascular, there was great pain, V = 0, enucleation.

The cornea was replaced by a slightly convex membranous structure about 1 *mm* in thickness, which consisted exclusively of granulation tissue, and which, becoming thinner at its periphery, passed directly over into the iris. On the posterior surface of this structure, with which the lens lay in contact, a thin layer of iris pigment could be distinguished.

The new-formed blood-vessels of the pseudo-cornea connect periphericaIly with the vessels of the iris. The sclera is thickened and infiltrated with leucocytes. The lens is top-shaped and cataractous anteriorly. The retina is detached and is only recognizable in its posterior third. It is thickened and infiltrated with leucocytes.

It is evident that here the original disease was a purulent retinitis through which the anterior two thirds of the retina were disorganized, while the posterior recognizable third still shows the typical picture of purulent retinitis.

The choroid is also recognizable in its posterior third only.

It is thickened, and the thickening increases as one passes forward from the optic disc. The epithelium is preserved, choriocapillaris and stratum vasculosum are distinguishable, and the supra-chorioidea as well ; the stroma is uniformly infiltrated with leucocytes.

There existed, then, a chronic inflammatory process in the greater portion of the choroid and in the ciliary body, which consisted chiefly in the development from the normal vessels of new-formed vessels, which passed into the inflammatory products or cellular masses about the choroid.

In this case also it cannot be doubted that after the injury it was the retina which played the leading rôle in the pathological process.

d.—CONTUSIO BULBI.

CASE 7 (Fig. 4).—Johann L., æt. thirty-six, struck in the right eye with the fist. On the following day the globe was swollen and injected, the vision reduced to the differentiation of light from darkness. He was admitted to the hospital on the twenty-third day.

The patient was a strong, healthy man, whose left eye was normal. The right eye had always been sound, and had good vision before the injury. The lids were now moderately swollen, the conjunctiva chemotic, the globe enlarged, prominent, and much retarded in its movements, the cornea slightly cloudy and containing some deep blood-vessels. The anterior chamber was abolished, no trace of iris could be seen, the somewhat cloudy lens lay adherent to the posterior surface of the cornea. There was an indistinct, yellowish-white reflex from the interior of the eye, V = o. The patient complained of unbearable pain, and desired the removal of the eye.

Enucleation performed on the twenty-fifth day. The injection of the enucleated globe was only partially successful. The ball was hardened in the ordinary manner and examined. The globe was enlarged.

Microscopic Examination (Fig. 4).—The cornea is infiltrated with leucocytes and contains new-formed vessels. The episcleral tissue, the thickened sclera, and the capsule of Tenon are all infiltrated with leucocytes. The retina is recognizable in its posterior half only, and shows here the changes characteristic of purulent retinitis. Farther forward it passes over into the purulent and cellular mass which fills the entire vitreous chamber. The separate layers of the choroid can be distinguished in its posterior half only, and it is here greatly thickened and its stroma infiltrated with leucocytes. Farther forward its limits are indistinct. Large new-formed vessels, originating in the normal blood-vessels of the choroid, pass into the cellular masses which fill the vitreous chamber.

The anatomical diagnosis is, then : retinitis purulenta ; choroiditis et cyclitis carnificans (fibrosa chronica) ; luxatio lentis in cameram anteriorem ; cataracta incipiens ; keratitis interstitialis inducta ; scleritis, tenonitis.

There could be no doubt in this case also that the purulent retinitis had played the important rôle and represented the anatomical basis of the clinical picture of panophthalmitis which was observed.

Case 8.—Ch., æt. thirty, left eye severely injured by a flail, hæmophthalmus, no open wound, total blindness, intense pain. I found the conjunctiva chemotic, the globe apparently enlarged, prominent, and having but slight motility. The cornea was diffusely opaque, and showed at its margin some deep blood-vessels. Anterior chamber shallow. The iris, as far as can be told through the cloudy cornea, is dislocated, the pupil blocked by exudative masses. The interior of the eye cannot be seen. Vision completely lost.

Enucleation on 6th day. Examination of the injected and hardened globe gave the following: Long diameter of globe 24 *mm*, transverse 23 *mm*. The cornea uniformly infiltrated with leucocytes, and having a few deep blood-vessels near its margin. The sclera and the capsule of Tenon also infiltrated with leucocytes. The retina in its posterior two thirds is clearly recognizable, shows here the same characteristics of purulent retinitis as the two cases preceding, and farther forward passes over gradually into the uniform purulent and cellular mass which fills the entire vitreous chamber.

The choroid is recognizable to the same extent as the retina, is moderately thickened and uniformly infiltrated with leucocytes.

The ciliary body is not detached, and is affected in a manner similar to the anterior portion of the choroid; small new-formed vessels pass from it through the pigment epithelium into the cellular mass. The iris is thickened and infiltrated with round cells.

e.—VULNUS CORNEÆ PERFORANS IN OCULO LÆSO.

CASE 9 (Fig. 5).—A., æt. forty-five, left eye, which had gradually become totally blind some years previously, was injured by a glass splinter a few days before his admission.

I found the bulbar conjunctiva slightly chemotic, the cornea irregularly curved and diffusely cloudy, having deep blood-vessels in places, and in the centre an obliquely running cicatrix 2½ *mm* long. The eye was somewhat exophthalmic, and could be moved but slightly. Severe pain. The eye was enucleated the following day, and injected and hardened in the usual manner.

Microscopic Examination (Fig. 5).—The globe is enlarged. The cornea is infiltrated with leucocytes, and shows deep blood-vessels, and in its central portion, cicatricial tissue. The membrane of Descemet is torn in the middle and wrinkled. The retina is considerably thickened, most markedly in the region of the optic disc, and exhibits the same pathological condition as was found in the first case with retinitis purulenta acuta traumatica. The pus cells lying on the surface of the retina are directly continuous with those which lie in the nerve-fibre layer. The optic disc is cupped. A glance at the preparation or the figure is sufficient to show that the purulent and cellular product which fills the vitreous chamber was derived chiefly, if not altogether, from the retina.

This was the freshest case of retinitis purulenta traumatica which showed clinically the picture of beginning panophthalmitis that I have had an opportunity of examining.

The choroid and the ciliary body are quite normal. The iris in its whole extent is adherent to the posterior surface of the cornea, and is thickened and infiltrated with leucocytes. A number of new-formed vessels from the blood-vessels of the iris pass into the exudative masses composed of fibrin and leucocytes which fill the enlarged posterior chamber.

It would be impossible for any one to doubt that in this case it was the retinitis purulenta alone which caused the clinical picture of beginning panophthalmitis. The active participation of the vitreous in the production of the purulent and cellular masses is with almost absolute certainty excluded.

Case 10.—N., æt. twelve, thrust a thorn through the centre of his left cornea, which had an old adherent leucoma. The patient was brought to me six days after the injury. The lids were swollen and red. The conjunctiva was chemotic, the globe apparently enlarged, somewhat prominent and immobile. In the centre of the cornea was a wound 4 *mm* in diameter not wholly cicatrized, about which the cornea shows a yellowish-white infiltration. The lens was cloudy, but a yellowish reflex could still be got from the interior of the eye. The vision was entirely abolished. On the 16th day the eye was enucleated.

The cornea contained leucocytes, deep vessels, and cicatricial tissue, which, passing through the ruptured membrane of Descemet and the capsule of the lens, united with the fibres of the latter (Fig. 6). Sclera and capsule of Tenon were infiltrated with leucocytes.

There was a funnel-shaped detachment of the retina. The posterior portion of the funnel forms a solid tumor-like mass, at the periphery of which both nuclear layers of the retina can be distinguished, while the remainder consists of granulation tissue (hyperplastic retinitis).

The closely crowded and rapidly proliferating leucocytes which lay on the inner surface of the granulation masses, as well as the budding blood-vessels found there, showed that these masses were in the stage of active growth at the time of enucleation.

There is no doubt that if the dioptric media had permitted a view into the interior of the eye, these masses would have simulated a tumor (pseudo-glioma). The vitreous chamber is filled with pus, cellular masses, and coagulated fibrin. The choroid, for the distance in which the retina it detached, is quite normal. Its anterior portion, to which the retina is still adherent, and the ciliary body are detached from the sclera, and send new-formed blood-vessels through the pigment epithelium and the pars ciliaris retinæ into the cellular masses filling the vitreous chamber.

The space between the choroid and the detached retina is filled with fibrin in whose meshes some few leucocytes are found.

CASE 12.—J., æt. eighteen, was received at my clinic October 13, 1889. As a child he had suffered from recurring inflammation of the eyes, which in the left had led to partial suppuration of the cornea, perforation, and the formation of a leucoma adherens. Some months before, perhaps in June, he had struck the eye against a piece of furniture and a violent inflammation ensued leading to total blindness.

The lids of the left eye are red and swollen, the bulbar conjunctiva chemotic. The ball is enlarged, protruding, and but slightly movable. The cornea is irregularly cloudy. A yellowish-white reflex is got from the interior of the eye.

Microscopic Examination.—The iris is adherent at one point to the posterior surface of the cornea. The anatomical diagnosis is: retinitis purulenta et hyperplastica incipiens ; choroiditis et cyclitis carnificans ; cicatrices corneæ ; keratitis inducta ; synechia iridis anterior ; cataracta incipiens ; scleritis ; tenonitis.

CASE 13 (Fig. 7).—K., æt. fourteen, had suffered from infancy with continuously recurrent inflammation of the eye, which had left opacities in the cornea. The left eye was wounded with a drill. The following day the ball and the lids were swollen, the vision was nearly lost, and the patient complained of violent pain. The patient being admitted on the twelfth day I found the lids red and swollen, the bulbar conjunctiva chemotic, the globe enlarged, prominent, and immobile. The cornea irregular in its curvature, cloudy, and vascular. A clear view of the interior was not gotten, but I seemed to get a whitish reflex from the depth of the eye. Vision totally abolished. Severe pain. The other eye showed slight cloudiness of the epithelium.

Microscopic Examination.—The cornea is irregularly curved and contains leucocytes, blood-vessels, and cicatricial tissue. At its margin there is a perforation through which a portion of the lens protrudes.

Sclera and Tenon's capsule are moderately infiltrated with leucocytes.

The retina, together with the choroid and ciliary body, is detached from the sclera.

The relations of the retina are similar to those of the case preceding. The posterior portion of the detached funnel has become converted into granulation tissue, and shows leucocytes in a state of proliferation, young connective tissue, and new-formed budding blood-vessels—*i. e.*, the picture of hyperplastic inflammation, while farther forward the walls of the funnel show clearly the retinal structure. The two nuclear layers are distinguishable ; the nerve-fibre layer, where it is still distinguishable, is covered with leucocytes, and passes over into the purulent and cellular masses filling the vitreous chamber without a perceptible line of demarcation.

The choroid is nearly normal in its entire extent and lies in apposition with the retina. A number of new-formed vessels pass from the ciliary body through the pigment epithelium and the pars ciliaris retinæ into the cellular masses of the vitreous chamber.

Panophthalmitis Secundaria e Keratitide Ulcerosa.

CASE 14 (Fig. 3).—N., æt. nineteen, suffered from a large osteo-sarcoma of the left shoulder. Four weeks before his admission an exophthalmus began which steadily progressed. I found the left eye prominent, dislocated outward and downward, and almost immobile. The dioptric media were clear ; the ophthalmoscope revealed a neuro-retinitis. $V = \frac{6}{8}$. Diplopia. Cerebral symptoms.

The clinical diagnosis was : sarcoma metastaticum cavi cranii et orbitæ sinistræ ; exophthalmus, neuro-retinitis oculi sinistri.

The exophthalmus and the displacement of the globe increased, and the cornea sloughed, after which panophthalmitis came on. The patient died.

The globe and the surrounding tissues were carefully injected and preserved.

Anatomical Examination.—The globe is elongated, 27 *mm* antero-posteriorly, 12 *mm* in lateral diameter, open anteriorly.

At the point of perforation the remainder of the cornea has united with the iris and formed a small spherical granuloma.

The detached retina is only recognizable in its posterior third. It is densely infiltrated with leucocytes and anteriorly it passes imperceptibly into the purulent masses which fill the whole interior of the ball.

The choroid and the ciliary body are detached from the sclera in most of their extent, and the suprachoroidal space is filled with pus, cellular masses, and coagulated fibrin. The chorio-capillaris is well preserved throughout, the stratum vasculosum is for the most part transformed into a purulent mass. The sclera is infiltrated with leucocytes, and considerably folded. About it, corresponding to the position of Tenon's capsule, are quantities of leucocytes and young connective tissue.

The ball is surrounded anteriorly by large hypertrophic folds, which correspond in position to the bulbar conjunctiva, the fornix, and in part the palpebral conjunctiva. These folds are covered with thickened epithelium, and consist of new connective tissue, new-formed blood-vessels, and leucocytes. We must consider these as being products of a hyperplastic inflammation of the conjunctiva. Upward and inward from the ball, sarcomatous masses are found.

We shall not be in error, if we assume that in this case retinitis and choroiditis purulenta developed simultaneously, and that both were factors in the production of the clinical picture of panophthalmitis. The presence of chemosis or conjunctivitis hyperplastica in this case seems to me noteworthy.

Atrophia Bulbi Progrescens e Panophthalmitide.

I shall mention but briefly several cases in which enucleation was performed after a panophthalmitis had run its course.

CASE 15.—J. R., æt. six, had suffered from frequently recurring inflammation of both eyes (kerato-conjunctivitis phlyctenulosa). April 1, 1876, one of these pustular phlyctenulæ led to a purulent infiltration and perforation of a considerable portion of the cornea, and a secondary panophthalmitis ensued, with enlargement, protrusion, and immobility of the globe, marked chemosis of the lids and conjunctiva, complete loss of vision and severe pain. A thick pus was poured out from time to time through the corneal perforation. The inflammatory symptoms decreased later.

The examination gave as the anatomical diagnosis: Retinitis purulenta; choroiditis purulenta et hyperplastica; iritis hyperplastica; scleritis, tenonitis.

In this case also it is probable that the pus filling the interior of the globe came from the retina, while that in the suprachoroidal space came from the stratum vasculosum of the choroid.

The three following cases correspond to that just reported in the most important particulars, both as to the clinical course and the anatomical changes found, and I shall therefore state them briefly:

Case 16.—J., æt. three, living in bad hygienic conditions in a damp cellar, suffered from kerato-conjunctivitis phlyctenulosa, which finally led to infiltration and perforation of the left cornea, and later to a secondary panophthalmitis. The eye was enucleated. The anatomical changes were nearly identical with those in the preceding case.

Case 17.—F., æt. four, was brought to my clinic with a broad marginal phlyctenula. The general condition was bad. The eye perforated and secondary panophthalmitis developed. The anatomical changes in the eye, enucleated after death, were similar to those in the preceding case.

Case 18.—K., æt. two, suffered from xerosis conjunctivæ infantilis, and his general condition was bad. Xerosis corneæ followed, then exfoliation, secondary panophthalmitis, and finally death. The anatomical changes here were similar to those in the preceding cases.

Panophthalmitis Metastatica.

I had at one time some sixteen cases of panophthalmitis metastatica, collected in the days before antiseptics were introduced into the maternity hospitals, when puerperal diseases were common, but these preparations have been taken surreptitiously from my collection, and I shall therefore only report briefly two recent cases from my clinic.

Case 19.—B., æt. forty-five, was admitted to my clinic eleven weeks after childbirth. The formerly enlarged eyeball was then small and but slightly movable, the conjunctiva somewhat chemotic, a yellowish-white reflex was obtained from the interior of the eye, the vision was entirely lost. Perforation of the sclera followed and further shrinking of the ball.

Microscopic Examination.—The sclera is thickened, folded in places, and infiltrated with leucocytes. The capsule of Tenon is likewise infiltrated. Retina and choroid are uniformly thickened and show tumor-like masses which consist of young connective tissue, new-formed vessels, and leucocytes. The small vitreous chamber which remains is filled with an exudative mass, which contains new-formed vessels, and which is in the process of becoming connective tissue. The iris is but slightly changed, showing some leucocytes. There are small masses of fibrin in the anterior chamber. The lens shows beginning cataract formation.

The question whether in this case the process developed originally from the retina or from the choroid is hard to decide, since the changes in the two membranes are so equally advanced.

CASE 20.—B., æt. twenty-four, was brought to my clinic Dec. 21, 1888, in a moribund condition without any history.

The eyes were moderately prominent and immobile, the conjunctiva chemotic, the interior of the eyes giving a yellowish reflex.

Diagnosis: choroiditis purulenta metastatica oculi utriusque.

The patient died some days later and the eyes were enucleated after death. I have preserved one, and given one to my assistant.

Anatomical Examination.—The globe is but slightly enlarged and of normal shape. The retina shows in its posterior portion the signs of retinitis purulenta, and its inner surface is covered with a thick layer of pus continuous with the leucocytes crowding the nerve-fibre layer, that are thickest about the vessels.

The choroid in its posterior portion, where the retinal affection is most marked, is thickened and infiltrated with leucocytes. Elsewhere it is normal, as are the ciliary body, iris, lens, and cornea.

Panophthalmitis after Various Injuries per Scleram.

CASE 21 (Fig. 8).—S., æt. forty-five, splinter of iron flew into the right eye. Two days later he was admitted to my clinic. I found the right lids red and swollen, the bulbar conjunctiva chemotic, the eyeball enlarged, exophthalmic, immobile, and in the sclera 5 *mm* outward from the corneal margin, a wound 2½ *mm* in diameter.

The cornea was steamy and the aqueous humor cloudy. The iris was discolored, the reflex from the interior of the eye intensely yellowish-white. Vision was abolished. Severe pain. The eye was enucleated on the fifth day.

Anatomical Examination.—Globe enlarged (Fig. 9), of obliquely oval form, long diameter 25 *mm*, transverse 27 *mm*. Cornea, sclera, and capsule of Tenon richly infiltrated with leucocytes. In the pars plana of the ciliary body there is a perforation through which pus exudes. The choroid is greatly thickened, and shows in its posterior portion, at the temporal side, large papillary excrescences. The choroid is infiltrated with pus and leucocytes in its whole extent, but most markedly just about the blood-vessels.

The retina has been entirely destroyed by suppuration. The foreign body, a splinter of iron 4 *mm* by 2 *mm*, lay in the purulent mass on the floor of the vitreous chamber.

This case was one of true fulminating choroiditis purulenta. It cannot be stated with certainty in what manner and when the retina was associated in the inflammatory process, but this probably occurred secondarily.

Case 22.—K., æt. sixty, a fragment of stone passed through the ciliary region into the interior of the eye. A typical case of panophthalmitis developed. Fifteen days later, when the violent symptoms had abated and the ball had again become movable, it was enucleated. The microscopic examination showed choroiditis purulenta and secondary retinitis purulenta.

Case 23.—N., æt. thirteen, stuck the blade of a penknife deep into the eye in the ciliary region. The typical picture of panophthalmitis followed. Enucleation four weeks later. The microscopic examination showed choroiditis purulenta and secondary retinitis purulenta.

Case 24.—K., æt. thirty-four, was stuck in the ciliary region by a fragment of a pot. The eye became blind and showed a cicatricial staphyloma of the cornea. Typical panophthalmitis followed. Enucleation ten days after the injury. The microscopic examination showed choroiditis purulenta. The entire retina had suppurated.

CASE 25.—P., æt. thirty-one, a splinter of iron, perforating the cornea, penetrated into the ciliary body, causing a typical case of panophthalmitis. The eye was enucleated, and the microscopic examination showed choroiditis purulenta with secondary involvement of the retina.

Recapitulation and Remarks.

Retinitis purulenta as the primary and chief lesion of the panophthalmitis was found in 10 cases, namely, in 2 of perforating wounds of the cornea (1, 2), in 3 of foreign bodies which had entered the eye through the cornea (3, 4, 5), in 1 of total burning of the cornea from melted iron (6), in 2 of severe contusion of the eye (7, 8), in 2 of injuries of the cornea in eyes previously diseased (9, 10).

Retinitis purulenta followed by retinitis hyperplastica was found in 3 cases (11, 12, 13).

Retinitis and choroiditis purulenta, in all probability coming on simultaneously, formed the anatomical basis of the panophthalmitis in 5 cases (14–18).

Retinitis purulenta was the lesion found in 2 cases of metastatic panophthalmitis after labor (19, 20).

Choroiditis purulenta was found in 3 cases in which foreign bodies had passed through the sclera in the ciliary region (21, 22, 25).

Choroiditis purulenta was found in two cases in which there had been penetrating wounds in the ciliary region (23, 24).

Leaving out of account the one case of metastatic panophthalmitis in which the primary disease could not be ascertained, there remain 24 cases of panophthalmitis, the anatomical examination of which showed the lesion to be undoubtedly retinitis purulenta in 11 cases (1–10, 20), probably retinitis purulenta in 3 cases (11, 12, 13), probably retinitis and choroiditis purulenta simultaneously in 5 cases (14–18), and a genuine choroiditis purulenta in 5 cases (21–25).

This shows that the clinical picture of panophthalmitis should not be assumed to depend always on a choroiditis purulenta, as is done in all text-books and other works and treatises on this disease, but that the clinical picture of typical panophthalmitis depends far more frequently on a retinitis purulenta than on a choroiditis purulenta.

Besides this principal conclusion which I have drawn from my studies, I would offer some others, although my material is too small to warrant me in considering these conclusions more than probabilities.

In every case in which a foreign body passed through the centre of the cornea into the interior of the eye, without wounding the uveal tract, the panophthalmitis which developed depended on a retinitis purulenta. This is also true of the cases of central perforating wounds of the cornea, of burning of the cornea, and of contusion of the ball where the blunt force reached the cornea from before.

In all cases of panophthalmitis secondaria inducta it is very probable that both retina and choroid were affected equally and at the same time by the purulent inflammation.

From this I would conclude that in secondary panophthalmitis caused by total suppuration of the cornea, whether after blennorrhœa, xerosis of the cornea, or severe phlyctenulæ, these two membranes are attacked by purulent inflammation at the same time, or one shortly after the other, and the lesion producing the symptoms of the panophthalmitis is in all probability a simultaneous retinitis and choroiditis purulenta.

Based upon the communications of Virchow (*Virchow's Archiv*, vol. ix., p. 307, and vol. x., p. 175), Nagel (*Græfe's Archiv*, vol. vi., 1, p. 219), Heiberg (*Med. Centralblatt*, No. 36, 1874), Oeller (*Archiv für Augenheilkunde*, vol. viii., p. 357), Hirschberg (the same *Archiv*, vol. ix., p. 299), and upon my own cases, I believe myself justified in drawing the probable conclusion that in *panophthalmitis metastatica*, the lesion is at times retinitis purulenta, at others choroiditis purulenta, finally perhaps both together.

In all my cases in which a foreign body passed through cornea and sclera into the interior of the eye with marked injury to the uveal tract, or where there were severe penetrating wounds in the ciliary region or farther back, choroiditis purulenta was found as the anatomical basis of the panophthalmitis.

A further result of my studies is the proof that so-called carnificating and hyperplastic inflammations almost invariably play an important part in the panophthalmitic process.

Choroiditis, cyclitis, iritis or retinitis carnificans seu chronica fibrosa is the process in which, from the normal vessels of these parts, numerous new-formed vessels develop, which run into the surrounding inflammatory products, which consist of cells and intervening substance.

In this way the exudative masses are organized, becoming young connective tissue, from which later cicatricial tissue may develop.

It is a process analogous to that in the lung, which has been described as pneumonia carnificans seu chronica fibrosa.

Choroiditis, cyclitis, iritis, retinitis, scleritis, conjunctivitis, tenonitis, etc., hyperplastica, on the contrary, is the process in which, through the exudation, proliferation, and further development of leucocytes, and through the development of new-formed blood-vessels in the part itself, there is formed an actual granulation tissue, and thus gradually a more or less marked hyperplasia of the part. These cells then develop into connective-tissue cells and may finally form cicatricial tissue.

In the first case there is an organization of exudative masses which lie on the part, in the second case there is a true hyperplastic thickening of the part itself.

Supplementary.

After this paper was concluded, I found by accident two eyeballs in my collection which had exhibited the clinical picture of panophthalmitis before their enucleation. Both of these eyes were enucleated at a late stage, when the panophthalmitis had nearly run its course, and since they are of interest as completing our picture of this pathological process, I shall describe them briefly.

CASE 26 (Fig. 9).—J., æt. twenty-six, was severely injured in the right eye by a bit of unslaked lime. The eye and lids swelled and were very painful. Sight abolished. Six days later I found a fulminating panophthalmitis. Three weeks later patient appeared again with beginning atrophy of the ball. The patient consented four weeks later to have the eye enucleated.

Anatomical Examination.—The cornea is replaced by a tumor-like mass, springing from the anterior portion of the uveal tract, consisting of granulation tissue covered with irregular epithelium, and containing leucocytes, young connective tissue, and a number of new-formed vessels.

The choroid is detached on the temporal side as far forward as the æquator of the eye, and completely detached on the nasal side together with the ciliary body. The entire choroid is degenerated into a thick layer of granulation tissue, consisting of leucocytes, young connective tissue, and new-formed blood-vessels, which is continuous on the temporal side with the tumor-like granulation tissue replacing the cornea.

We see in this case, as in none of the foregoing, how the new-formed blood-vessels from the detached choroid develop in two directions, inward and outward.

Case 27.—D., æt. twenty-six. A fragment of steel about ten *mm* in diameter pierced his left upper lid and remained fast in the cornea and sclera. Removed, it left a wound ten *mm* long, which extended from the centre of the cornea downward and inward through the ciliary region into the sclera. The anterior chamber and the conjunctival sac contained blood. The vision was abolished, and the patient complained of severe pain. In a week the typical picture of fulminating panophthalmitis developed. Two weeks later I removed the shrinking globe.

Anatomical Examination.—The cornea is replaced by a large fungus-shaped mass of granulation tissue and young connective tissue springing from the uveal tract. The conjunctiva is irregularly thickened and contained leucocytes, young connective tissue, and new-formed blood-vessels. The sclera is thickened and infiltrated with leucocytes, as is the capsule of Tenon. Retina and choroid, recognizable in their posterior portion only, pass over, farther forward, into the general granulation tissue.

If we take now a general oversight of the entire panophthalmitic process, we find that, whether the disease be of traumatic, secondary, or metastatic origin, it commences constantly with a fulminating purulent retinitis or choroiditis, or with both together. To this is soon added a scleritis and tenonitis, and, if the cornea has not been previously destroyed, a keratitis inducta interstitialis follows, which may assume a suppurative character later. Following this come the carnificating and hyperplastic inflammations of the various parts of the globe, particularly of the uvea. In general the carnificating processes precede the hyperplastic, so that the original purulent masses are gradually replaced by granulation tissue, from which later young connective tissue develops, filling at last the whole interior of the globe. Finally the new-formed connective-tissue masses may undergo a cicatricial shrinking, and retrograde metamorphoses take place.

EXPLANATION OF THE FIGURES.

Fig 1 shows a longitudinal section of the eye of Case 1, enucleated for panophthalmitis after a central penetrating wound of the cornea. The pear-shaped globe, open anteriorly, is filled with pus. The remaining portion of the retina exhibits the picture of purulent retinitis ; the entire uveal tract, that of hyperplastic inflammation in the first stages.

The tongue-formed prolongations growing from the capsule of Tenon into the sclera are to be considered as a tenonitis hyperplastica.

Fig. 2 represents a longitudinal section of the eye of Case 6, with panophthalmitis after combustion of the cornea with melted iron. Anteriorly we see a membrane composed of granulation tissue replacing the cornea, which is to be considered as a product of iritis hyperplastica. The vitreous chamber is filled with pus and cellular masses. The retina, only recognizable in its posterior portion, shows the picture of purulent retinitis ; choroid and ciliary body show the picture of carnificating inflammation.

Fig. 3 represents a section of the eye in Case 14, of secondary panophthalmitis in consequence of xerotic suppuration of the cornea. The globe, open anteriorly and stretched in an antero-posterior direction, is surrounded by folds of hyperplastic conjunctiva (conjunctivitis hyperplastica), and its interior is filled with pus. Retina and choroid show the picture of purulent inflammation.

Fig. 4 shows a section of the eye in Case 6, having panophthalmitis in consequence of contusion of the ball. The lens, escaped from its capsule, is luxated into the anterior chamber, the iris being drawn together behind it; the periphery of the vitreous is filled with pus and cellular masses. Retinitis purulenta; choroiditis and cyclitis carnificans; keratitis inducta; scleritis, tenonitis, and cataracta incipiens.

Fig. 5 represents a section of the eye in Case 9, of beginning panophthalmitis after perforating corneal wound in a weak-sighted eye. It shows the characteristic picture of retinitis purulenta and iritis carnificans. The iris lies in apposition with the cornea; choroid and ciliary body are almost normal; the cornea contains leucocytes, cicatricial tissue, and deep blood-vessels.

Fig. 6 represents a section of the eye in Case 11, of panophthalmitis after injury of the previously diseased eye. The lens is adherent to the cornea; synechia lentis; the posterior half of the retina is detached. Retinitis hyperplastica; choroiditis and cyclitis carnificans; amotio corporis ciliaris; cicatrices corneæ; keratitis inducta; cataracta incipiens.

Fig. 7 shows a section of the eye in Case 13; also panophthalmits after a penetrating corneal wound in an eye previously diseased. Prolapse of the lens, phacocele. Retina and choroid detached. Retinitis hyperplastica; cyclitis carnificans; cicatrices corneæ; keratitis inducta; cataracta incipiens.

Fig 8 shows a section of the eye in Case 21, of fulminating panophthalmitis after the passage of a splinter of iron through the sclera into the vitreous chamber. The sclera on the left side ruptured and wrinkled, the entire vitreous chamber filled with pus. The choroid shows the typical picture of a purulent choroiditis; no trace of the retina can be seen. Keratitis inducta, scleritis, tenonitis.

Fig. 9 represents a section of the eye in Case 26, with beginning progressive atrophy after panophthalmitis in consequence of burning with unslaked lime. Uveitis totalis hyperplastica with simultaneous choroiditis carnificans. Retinitis hyperplastica, scleritis, tenonitis. In the centre of the globe a small localized collection of pus.

INSUFFICIENCY OF THE OBLIQUE MUSCLES.

By G. C. SAVAGE, M.D.,

PROFESSOR OF OPHTHALMOLOGY IN THE MEDICAL DEPARTMENT OF VANDERBILT UNIVERSITY,
NASHVILLE, TENNESSEE.

(*With five drawings.*)

EVERY ophthalmic surgeon, however skilled in correcting errors of refraction and in operating for the different known forms of heterophoria, has had cases of eye-strain for which he could do but little. In investigating a few such cases during the last six months I have found the cause to be a want of equilibrium on the part of the *oblique muscles*. The detection of this condition is easy. I place a double prism (my modification of the Maddox prism) before one eye, the other for the moment being covered, and ask the patient to look at a horizontal line on a card held eighteen inches away. The effect of the double prism (each 6°, bases in), so placed that the axis is vertical, is to make the line appear to be two, each parallel with the other. The other eye is now uncovered, and a third line is seen between the other two, with which it should be perfectly parallel.

While a change of the position of the axis of the double prism from the vertical towards the horizontal will alter the distance between the lines, their direction will be unchanged, hence no loss of parallelism. This fact admits of a little carelessness in the placing of the prism in the trial-frames, though the axis should be vertical so as to give the maximum distance between the two extreme lines.

If there is a want of harmony on the part of the oblique muscles this test will show it at once in a want of parallelism of the middle with the two other lines, the right end of the middle line pointing towards the bottom and the left end towards the top line, or *vice versa*, depending on the nature of the individual case.

Fig. 1.

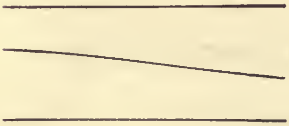


Fig. 3.

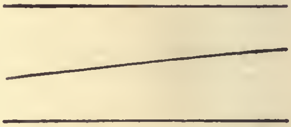


Fig. 2.

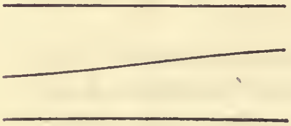


Fig 4.

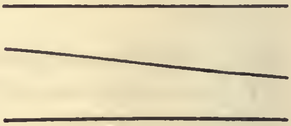
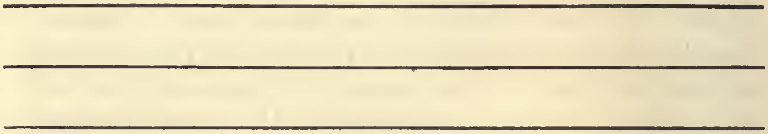


Fig. 5.



In my investigations I have always considered the eye before which no prism is held as the one under test. With the double prism before the right eye the patient is asked about the position and direction of the middle line. It may be nearer the bottom, thus showing left hyperphoria; or, again, it may extend farther to the right than the other two and not so far to the left, thus showing exophoria, or *vice versa*, showing esophoria. If the right ends of the middle and bottom lines converge while the left ends diverge, the superior oblique of the left eye is at once shown to be in a state of underaction. Fig. 1 represents such a test of the left eye. Fig. 2 shows a test of the left eye when the inferior oblique is the too weak muscle. Fig. 3 represents a test of the right eye, the loss of parallelism between the lines being due to underaction of its superior oblique, and Fig. 4 the same condition of the inferior oblique of the right eye. Fig. 5 represents a test of both eyes when there is perfect equilibrium of the oblique muscles.

As is well known, the function of the oblique muscles is to keep the naturally vertical meridians of the two corneæ parallel even when not vertical. This must be, or a troublesome form of double vision will result. If there is perfect equilibrium of the obliques, this parallelism of the meridians named is maintained without trouble; but if the superior oblique of either eye be too strong for its inferior, or *vice versa*, the parallelism of the vertical meridians is preserved, and double vision prevented only by excessive work on the part of the weaker muscle. This condition of the oblique muscles brings on, at longer or shorter intervals, a train of nervous symptoms for which, at present, I can see no hope of prevention or cure.

A CASE OF CONGENITAL DISPLACEMENT OF BOTH PUPILS.

By W. E. BAXTER, M.D., Bangor, Me.

Mr. M., American, aged fifty-two, consulted me August 8, 1890, complaining of severe pain in the eyes. He states that he has always been "near-sighted," and that people have frequently told him that his eyes were *queer*.

Three years ago, while cutting wood, he was struck on the left eye with a splinter. The eye and lid became very much inflamed, but in a week or ten days this passed away. He then noticed that he could not see as well as before. One year after this he was struck in the same way in the right eye, but this did not seem to cause him any trouble. For the last month pain has been very severe, starting in the left eye, and now also affecting the right eye. He had no pain until a month ago.

On examination the following condition was found: V O D $\frac{1}{60}$, with $+ 10$ D $\frac{5}{60}$. V O S, fingers at .5 *m*. No improvement with glasses. Right eye, T_n; left, $+T_1$. Pupils of both eyes displaced to the upper and outer edge of the cornea. Irides blue, right *very tremulous*, left muddy; in the left anterior chamber the small dark lens is visible; the irides forming a curtain as it were with only a small opening at upper part, through which the red reflex is seen, but no vessels or edge of the lens in the right eye.

No treatment was attempted, as the patient refused the proposed removal of the dislocated lens, with an iridectomy for optical purposes.

THE PERFECTED PRISMOMETER: ITS PRACTICAL ADVANTAGES, CONSTRUCTION, AND VARIOUS APPLICATIONS.

By CHAS. F. PRENTICE, New York.

(*With ten woodcuts.*)

IN the first numbers of these ARCHIVES of the year 1890, I described "A Metric System of Numbering and Measuring Prisms," which represented the result of a careful and extensive study of the subject, due to the suggestion of Dr. S. M. Burnett, who had entrusted me with the problem of searching for a system which should prove satisfactory to ophthalmologists as well as avoid conflict with the practical methods of manufacturing opticians. At the close of my investigations I felt that this had not only been accomplished, but that also an instrument in support of that system had been offered as a valuable assistant to opticians.

My familiarity with the routine of manufacture would not allow me to lose sight of the practical side, so that this, being a matter of primary importance to opticians, was kept well in view from the outset. In advocating the metric system and the use of the prismometer, I shall therefore here only do so in so far as they relate to the interests of manufacturing and dispensing opticians; the advantages of the system to ophthalmic practice having been previously set forth.

My argument in favor of the metric system **was** and **is** based upon the **unavoidable variability in the angles of our prisms,** and which must result from the present process of manufacture. Although this has been indicated in the previous papers,[1] I shall here take the liberty of quoting from a paper read in connection with my exhibit of the prismometer before the New York Academy of Medicine, October 20, 1890:

"It would, however, be exceedingly difficult and correspondingly expensive to manufacture prisms producing only fixed intervals of deflection. To render prisms sufficiently inexpensive as spectacle glasses it is necessary that they should be produced in large quantities at one grinding."

"The process at present consists in fastening a number of slabs of glass, by means of pitch, or other resinous material, upon a metallic surface-tool. The friction in grinding generates more or less heat, which at times is sufficient to soften the pitch and cause it to yield beneath the slabs. Some slabs will shift more than others, so that the prism-angles will vary more or less throughout. Besides, the underlying layer of pitch can never be of a uniform thickness." Were it not for these facts, I believe competition alone would long ere this have resulted in greater uniformity.

By means of the prismometer I found prisms, more especially of low degree, to vary between ten per cent. and thirty per cent. of their indicated numbering.

It is obvious that if manufacturers were obliged to discard all those prisms which varied from desired fixed intervals of prism-angle, **minimum** deviation, or any other designated deflection, the price would have to be increased on the perfect prisms sufficiently to compensate for the cost of those rejected, and which would have consumed equally as much material, time, and labor to produce.

Without confining myself to the deflections which should, by calculation, correspond to the prism-angles and index, I found, by means of the prismometer, among a series of prisms, of best Parisian manufacture, only the following number to produce deflections which were *even* alike:

Three doz. prisms	1°	2°	3°	4°	5°	
Number alike	6 = 1.1	7 = 2	6 = 3.1	6 = 3.7	8 = 4.6	prism-dioptries
Balance varying between	0.8 & 1.6	1.8 & 2.5	2.6 & 3.2	3.4 & 3.9	4.4 & 4.8	" "

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[1] ARCHIVES OF OPHTH., xix., Nos. 1 and 2, 1890.

These prisms were taken from original papers, and may be credited with having been made of the same material, at the same time, and upon the same tools. Greater precaution on the part of the manufacturer could not be expected.

To the *careful* reader of my papers it must have been apparent that **stress** had **nowhere** been laid upon the possibility of a variability in the index, but, on the contrary, that all my deductions were referred to the commonly accepted index of 1.53.

The privileges, however, were mentioned[1] which manufacturers might avail themselves of, both in respect to prism-angle and index, in seeking to provide prisms of the desired properties.

To any one familiar with the use of optical theodolites[2] and spectrometers[3] it must further be apparent that an endeavor to measure the *minimum* deviation, with prisms of small angles especially, is very tedious and difficult. The apparatus is expensive, requires a degree of accuracy in manipulation, and a knowledge in the reading of verniers, with which opticians can not readily be made familiar. To mount such prisms accurately upon the table of the spectrometer, and rectify the various adjustments of the instrument, are tiresome and slow operations which alone are sufficient to condemn its daily use by opticians whose work must necessarily be expeditious. In the physical laboratory, however, the instrument is undoubtedly invaluable. If the use of an instrument is to be abandoned for measurement of the minimum deviation, we shall find that manufacturers will simply divide the prism-angles by two (2), for the new nomenclature, and so give us the old culprit disguised under a new name. There would be great commercial convenience to be sure, in being able to dispose of the same prism under two names, but no *reform* in the interest of scientific exactness could be effected *without* measurement. Will it be policy under such circumstances to adhere to the *minimum* deviation merely for principle's sake? As the prismometer is intended to measure the refraction of prisms, in terms of the prism-dioptry, it may be well, for the benefit of those who may have found its **simplicity** obscured by the mathematical portion of my papers, again to explain its principles in more simple and somewhat different terms.

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[1] ARCHIVES OF OPHTH., xix., No. 1, p. 67, 1890.

[2] *Lehrbuch der Physik.*, Prof. Joh. Müller, Braunschweig, 1878.

[3] "Practical Physics," Glazebrook & Shaw, London, 1889.

"Elements of Physical Manipulation," Prof. Ed. C. Pickering, Boston, 1873.

We know that a lens-dioptry is the unit of refraction, and corresponds to a lens of one meter focus, Fig. 1.

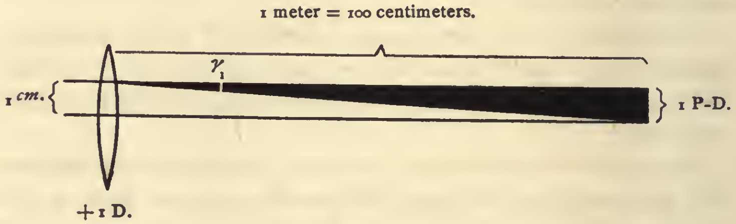

FIG. 1.

The prism-dioptry, *since lenses are but a fusion of prisms of varying angle*, may then be said to be the **linear deflection** which the refracted ray sustains **at the focus of a meter-lens,** when the incident ray impinges upon a peripheral portion of the lens **one centimeter** from the optical centre (Fig. 1).

The prism-dioptry therefore also represents the measure of the angle of deviation γ_1, for an eccentricity or decentration of one centimeter (Fig. 1).

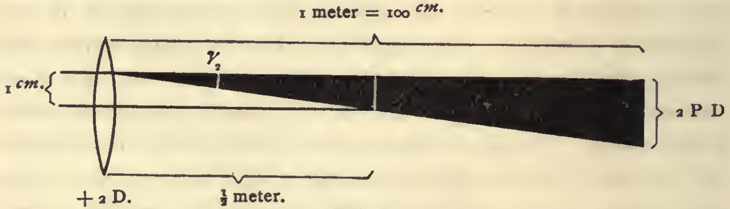

FIG. 2.

A ray impinging upon the same point of a 2-dioptry lens (Fig. 2) will sustain the *same unit* deflection at its focus ½ meter, and will therefore find the measure of its angle of deviation γ_2, expressed by twice the deflection at the meter-plane, or 2 prism-dioptries. A lens being decentred twice or half as much will produce twice or half as many prism-dioptries as the lens possesses lenticular dioptries of refraction.[1] The prism-dioptry is therefore but a sequence to the lens-dioptry. Nothing can be more simple. Thus the prism-dioptry represents the proportion 1:100, which is expressive of a grade of angular inclination in **daily use** by engineers and scientists the world over. To reduce prism-dioptries to degrees of angular deviation, it is only necessary to divide the prism-dioptries by 100, when they will represent the tangents to correlative angles in degrees, which are to be readily found in any table of sines and tangents. Since different lenses, through varying decentration, will produce different values of the angles of deviation $\gamma_1 \gamma_2 \ldots$, how will it be possible to determine the value of such angles in degrees, minutes, and seconds? The instrument is yet to be invented. The prism-dioptry and the prismometer solve the problem, and in a manner simple and rapid enough to any one of ordinary intelligence.

Since the therapeutic value of prisms is conceded, and their combination with lenses in practice frequent, the prismometer has been constructed with due regard to such combinations, making it possible by its aid to utilize to advantage the prismatic action due to decentration of the lens, for the purpose of offsetting the error which *invariably* exists in the constant prism, after the combination has been ground. Would it not then seem unwise and even arbitrary to hamper the dispensing optician in the practical fulfilment of his work by forcing him to a system of degrees, merely because it harmonizes with the designation of a strabismus which is incorrigible by prisms, or with the graduations found upon perimeters, ophthalmometers, etc., which have no connection with prisms whatever?

The metric system certainly possesses the commendation of reducing all the glasses of the trial case to a uniform nomenclature in dioptries. This alone should be considered a *practical* advantage, fully offsetting the merits of an *unverified theoretical minimum* deviation.

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[1] "A Metric System of Numbering and Measuring Prisms," ARCHIVES OF OPHTH., xix., Nos. 2 and 3, 1890.

If I may be permitted to offer a suggestion, let us learn to comprehend the power of our prisms by the limits of refraction, shown by the solid triangles in the preceding figures, when it will become wonderfully easy to fit these into meter-angles, or for that matter to any other angles in space, without necessarily confounding prism-dioptries with meter-angles, or meter-angles with "deviations of the eyes in height," as stated by Dr. Landolt.[1] The latter mistake could only be the result of a misconception of the definitions of the prism-dioptry and its relations to the meter-angle.

In recommending the metric system to the profession and practical opticians, I in conclusion beg to call attention to its superior advantages, as follows:

1. From a mechanical point of view, by taking the unavoidable difficulties of manufacture into consideration.
2. From a commercial and pecuniary point of view, by avoiding unnecessary expense in the production of prisms.
3. By the prismometer, which enables opticians to accurately fill the demands of the system.

Any system which neglects these important considerations *cannot* be considered progressive, nor can it effect a *reform* in the present necessarily haphazard endeavors of the dispensing optician, with whom so much of the blame and responsibility must rest. Opticians have always been on the alert to meet the requirements of the profession, and will no doubt gladly avail themselves of a system and an instrument which will enable them to sustain their reputations as mechanicians.

Taking all the facts into consideration, it suffices to say, that we have prisms of almost every imaginable deflection on hand in the market to-day, so that it merely requires an instrument of simple construction, which may be used in making the proper selection with **accuracy** and **despatch**, and this is precisely what is claimed for the prismometer, which it is my purpose here to describe.

In the accompanying illustration, Fig. 3, the essential operative parts of the instrument are shown as being mounted upon a triangular truss which is pivoted by a suitable joint to a pedestal, so as to permit of convenient inclination of the whole.

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[1] "On the Numbering of Prismatic Glasses," ARCHIVES OF OPHTH., xix., No. 4, 1890.

The graduated bar is rigidly supported near its extremities, upon the truss, by two short studs or pillars, the latter being slightly higher than the radius of the circular stage, which is supported at its back by a rod, fitted, sliding, and acted upon by a spring within the bar, so as to automatically effect contact of the face of the stage with the knife-edge, which is also mounted upon the truss, between the stage and the pinhole eye-piece.

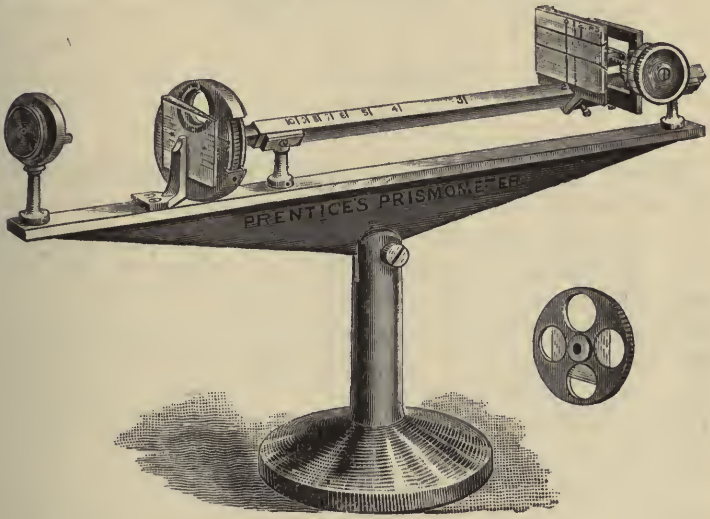

FIG. 3.

The divisions of the graduated bar, numbered 2, 3, 4 up to 10, are placed at $\frac{1}{2}$, $\frac{1}{3}$, $\frac{1}{4}$, up to $\frac{1}{10}$ of the meter[1] length, counted from the knife-edge, which represents the zero-end of the scale. A plane, perpendicular and arranged to slide upon the graduated bar, termed the index-plate, is provided with the index-line, marked zero (0), and two graduations at the right-hand upper edge, marked 1 and 2, which, being equal to correlative centimeter deflections at the meter-plane, correspond to their equivalents in prism-dioptries.

To facilitate subdivision of these graduations the index-plate is provided with a transverse slide, bearing its allotted part of the index-line, which is rendered adjustable by a milled head and micrometer screw, the first complete rotation of which will cause this section of the index-line to travel from 0 to 1, the second complete rotation taking it from 1 to 2. The milled head, being divided into 100 parts, enables us, by its graduations, to determine the position of the index-line of the transverse slide, relatively to the graduations upon the face of the index-plate, in 10ths and 100ths.

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[1] It has been found convenient to construct the instrument to half scale throughout.

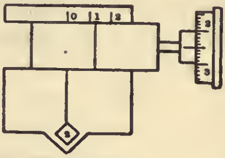

FIG. 4.

Thus, in the accompanying figure (4) we read from the face of the index-plate "1" and from the milled head $\frac{2}{10}$ths and $\frac{5}{100}$ths or 1.25 for the position of the index-line of the transverse slide.

As all readings of deflection must be reduced to the *meter-plane*, it will be necessary to note the position of the index-plate, *which must at all times correspond to one of the graduations of the bar*. Consequently, if the above reading is taken from the index-plate, when placed at the figure "2" of the bar, we shall have twice the number of prism-dioptries at the meter-plane, or $1.25 \times 2 = 2.5$ P-D.

For a reading of "2," from the index-plate, when placed at the graduation upon the bar marked "10," we have 20 P-D, which is the maximum measuring capacity of the instrument. In other words, it is merely necessary to *multiply* the readings of the index-plate by that figure upon the bar which defines the position of the index-plate upon it.

Before placing a prism in position for measurement, it is necessary to carefully determine its *passive* or non-refracting plane. This is accomplished by such slight rotary adjustment of it before the eye, until a line, situated at a convenient distance, is sighted as an unbroken one, being precisely the same method which we employ in determining the axes of cylinders. For convenience of registration, ink dots, in collimation with said line, should be applied to the prism. The stage is provided with a series of horizontal lines, engraved upon it, to facilitate perfect adjustment of the base-apex line of the prism, which is to be introduced between the stage and the knife-edge, with its apex to the right, and gradually forced downward while the ink dots pass successively from one horizontal line of the stage to the other, until the upper edge of the prism exactly bisects the circular opening in the stage. In this position the prism will exactly cover the lower half of the opening, while its lateral upper edge will be in collimation with the lower edge of the transverse slide. On completion of this adjustment it is of the utmost importance that the ink dots should coincide with one of the parallel lines of the stage. The observer's eye being placed before the eye-piece, will now perceive the upper edge of the index-plate, and the index-line at zero of the transverse slide, in their true positions, whereas the lower portion of the index-plate, with its index-line, being seen through the prism below, will appear displaced to the right. The position of the observer's eye is now to be carefully maintained, while the graduated milled head is operated with the right hand, until the index-line of the transverse slide has been shifted sufficiently to the right to make contact with the lower index-line seen through the prism. Perfect coincidence of these lines is necessary for an accurate determination of the deflecting power of the prism at any distance. It will consequently be well to previously remove any roughness of the upper base-apex edge of the prism by grinding it to a flat dull edge, and, to be very precise, to take the mean of several readings while the prism is in an undisturbed position. As an example, we shall suppose the prism to have been carefully adjusted in the manner described, and that our readings for three positions upon the bar from the index-plate are as follows :

2d Graduation of the bar, index-reading = 1.57 × 2 = 3.14 P-D.
3d “ “ “ “ “ = 1.05 × 3 = 3.15 “
4th “ “ “ “ “ = 0.78 × 4 = 3.12 “

$$\text{Mean : } \frac{9.41}{3} = 3.13 +$$

This precaution, in the interest of exactness, may appear to be unnecessary to some, yet it is here introduced as an exhibit in favor of the capabilities of the instrument.

The prismometer is particularly valuable when it is desired to measure the inherent prismatic action of decentred lenses, and their combinations with prisms.

In such cases it will be necessary to remove a peripheral portion of the lens by grinding it to a dull flat edge, as shown in the accompanying Figure 5.

The lens is then to be placed upon the stage with the flattened edge up, so as to cover half the stage opening ; the index-line of the transverse slide having been previously adjusted to zero (o). If, in sighting through the eye-piece, the index-line appears disjoined, it will only be necessary to shift the lens slightly to the right or left to re-establish coincidence of the lines, when the lens is said to be *centred.* While in this position ink dots should be placed upon the outer edges of the lens over a centrally situated horizontal line of the stage, as shown. For this centred position of the lens, in sighting through the eye-piece, we shall find the index-line at zero (o) unbroken, while the lower half of the index-plate will be enlarged or diminished according to the character of the lens employed. Supposing the lens be 3 D convex, we shall find the index-plate to present this view (Fig. 6) when it is placed at the graduation marked " 3 " upon the bar.

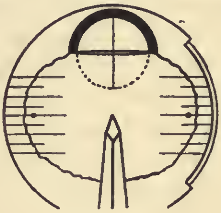

FIG. 5.

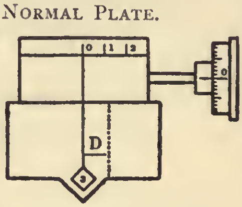

FIG. 6.—MAGNIFIED PLATE.

The lower half of the index-plate is provided with a red line, indicated by a *dotted* line in the figure, corresponding to a deflection of 1 P-D, and which appears proportionately *magnified.* As it will be inadmissible, in our readings, to place a magnified scale on a par with the normal scale of the prismometer, it will be necessary to *register* the magnified unit upon the upper portion of the index-plate, for reference and comparison during decentration of the lens. To accomplish this we displace the index-line of the transverse slide until it coincides with the red line (dotted line, Fig. 7), which, as far as the lens is concerned, now *represents* and *takes the place of* 1 P-D on the index-plate.

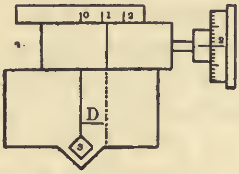

FIG. 7.

Now, by slowly shifting the lens to the left, we shall observe the lines of the lower index-plate to shift to right (Fig. 8).

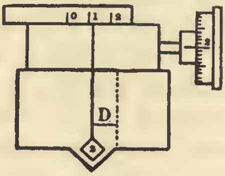

FIG. 8.

When the 3-D lens has been decentred one centimeter, experiment shows that the lower black index-line cuts the index-line of the transverse slide above. Bearing well in mind that the position of the *upper* index-line now *represents* "1," and that our reading has been taken for a position of the index-plate upon the bar at "3," we have 3 P-D as the result of decentring a 3-D lens one centimeter.[1]

In case, however, that the refraction of the lens as well as its decentration have not been previously determined it will be necessary to note the following (see Fig. 6):

It is evident that the $\frac{\text{normal plate}}{\text{magnified plate}}$ as $\frac{1}{D}$, so that the normal prism-dioptries sought $= \frac{1}{D} \times$ magnified readings, for convex lenses, which will be when $D > 1$, and

" " " $= \frac{1}{D} \times$ diminished readings, for concave lenses, which will be when $D < 1$.

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[1] "A Metric System of Numbering and Measuring Prisms," ARCHIVES OF OPHTH., xix., Nos. 2 and 3, 1890.

It is therefore only necessary to divide the magnified or diminished readings by D.

The value of D, as we have seen, is determined by first *centring* the lens. It will have a different value for different lenses, and will depend upon the distance of the lens from the index-plate. In fact, D represents the magnifying or diminishing power of lenses for any position of an object, when viewed through them, and which may be placed within their respective focal distances. For the 3-D convex lens, at the graduation upon the bar marked "3," measurement by the instrument shows D to be equal to 1.2, Fig. 7. Suppose we decentre a + 3-D lens until we obtain a reading say of 0.6 P-D, which is of course a *magnified* reading, we then have $\frac{0.6}{D} = \frac{0.6 \text{ P-D}}{1.2}$ (magnified reading) = 0.5 normal P-D at the distance "3," or 1.5 normal P-D at the meter-plane.

In measuring sphero-prismatic lenses we shall therefore find that the value of the constant prism can either be increased or diminished by a decentration of the lenticular element of the combination, a decentration of 5 *mm* in the above instance being sufficient to contribute 1.5 P-D. ad- or ab-ductive as occasion may demand.

By such means it will be possible to *counteract* the inaccuracies which *invariably* exist in the constant prism after the combination has been ground. When the lens is combined with a prism the flattened dull edge should be cut parallel with the true base-apex line, the latter being registered with ink dots and adjusted upon the stage as usual.

The most ready means of measuring such a combination—for example, + 3 D spherical combined with 2 P-D (constant prism) — will be to place the index-plate at the distance upon the bar marked "3," when, as before, the lens magnification D = 1.2, and which may be more conveniently determined by *previously centring* a spherical lens of the same refraction. Now, by deductive reasoning, we know that 2 *normal* prism-dioptries will be equal to $\frac{2}{3}$ P-D at $\frac{1}{3}$ the distance, and this would require to be 1.2 *greater* at the *same* distance to appear as the properly proportioned magnified deflection seen through the lens, consequently $\frac{2}{3}$ 1.2 = 0.8 magnified prism-dioptries. We therefore set the line of the transverse slide so as to read 0.8 P-D at the distance marked three ("3") upon the bar, and proceed to decentre the lens until the lower index-line cuts it, when we shall have the desired 2 *normal* prism-dioptries. We may utilize the rule to prove the result: 0.8 mag. P-D $\times 3 = \frac{2.4}{D} = \frac{2.4}{1.2} = 2$ *normal* P-D. Since lenses are capable of providing as many prism-dioptries as they possess lens-dioptries of refraction, it also follows that we shall occasionally be enabled to secure a considerable proportion of prismatic action by decentration alone, provided the spherical lens is of proportionately greater strength. For instance, the 3-D lens will produce 3 P-D for a decentration of $1\,\underline{cm.}$ so that an available decentration of $3\frac{1}{3}\ {}^m/_m$ could in itself be relied upon to furnish 1 P-D of the 2 P-D in the lens forming the subject of our example.

To facilitate measurement of concave sphero-prismatic lenses the stage is provided with a rotating disk, within, containing three prisms of varying power, with their bases down, and which may be successively carried before the lower half of the opening in the stage as occasion may demand.

The object of these prisms is to counteract the prismatic action in the vertical plane, which would otherwise manifest itself by a confusion of the transverse slide in its contact with the lower portion of the index-plate (Fig. 9), as a result of sighting through the upper peripheral edge of a concave lens (acting as a prism with its base up) when placed in proper position on the stage. The extent of the confusion of the parts, as shown in the figure, will naturally depend upon the strength of the lens, so that rotation of the disk will reveal the prism best calculated to re-establish contact, as shown in Fig. 10.

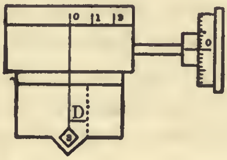

FIG. 9.

Our choice of the prism being made, the lens is to be removed from the stage so as to *rectify* the position of the disk-prism before the index-line at zero (0), which should naturally present a *perfect* vertical line to view.

As an example, let us suppose the combination — 3 sph. $\overset{\frown}{\smile}$ 2 P-D (constant prism) to be presented for measurement. We should first select a concave 3-D lens, centring it upon the stage as described, and discover a confusion of the index-plate, at "3" upon the bar, as shown in Fig. 9.

It will be found that the first prism of the disk proves sufficient to re-establish contact, as in Fig. 10.

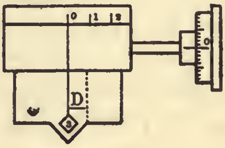

FIG. 10.

Removing the lens, *rectifying* the disk-prism, and *replacing* the lens, we find the diminishing power of the lens D = 0.83. Our object being to secure 2 P-D at the meter-plane, it follows that $\frac{1}{3}$ of this will have to be the reading from the index-plate at "3" upon the bar, or $\frac{2\ \text{P-D}}{3} = 0.67$ in the absence of diminishing power, and consequently $0.67 \times 0.83 = 0.55$ as a result of diminution by the lens.

The index-line of the transverse slide is therefore to be set to 0.55. The spherical lens is now to be replaced by the sphero-prismatic lens, with its base-apex line marked and adjusted upon one of the horizontals of the stage, and shifted upon this to the right or left, until the lower index line cuts the index line of the transverse slide. While the sphero-prismatic lens is in this position, an ink dot is to be placed upon it at the knife-edge, as the dot is intended to ultimately occupy the centre of the frame in which the lens is to be mounted.

Such can be the accuracy of the optician's work, with the aid of the prismometer for the metric system, and of which oculists in America may readily avail themselves by a simple request to have their diagnostic prisms re-numbered by measurement upon the instrument. By these explanations I hope to have succeeded in conveying the fact, that my object has not only been to promulgate a *theory*, but also to render it *useful* and fully *subservient* to *practice*, in the absence of which it should, like many another, only live in minds, and mould in books.

ORBITAL OPTIC NEURITIS, INCLUDING ALCOHOL AND TOBACCO AMAUROSIS.[1]

By H. KNAPP, M.D.

THE singular cases in which people notice a haze, a blurr, even a perfectly dark patch, in the centre of their field of vision, known under the name of central amblyopia or central scotoma, as well as those cases in which persons lose their sight in one or a few days without exhibiting symptoms of any ocular or general disease to account for this appalling occurrence, were, more than twenty years ago, accounted for by A. v. Graefe[2] and Theo. Leber[3] by the supposition of an inflammation in the orbital part of the optic nerve, **retrobulbar neuritis.** By anatomical investigations during the last ten years, this hypothesis has been proved to be a reality, as we shall see further on.

Retrobulbar neuritis may be idiopathic or the result of different kinds of intoxication, alcoholism, nicotinism, lead-poisoning, diabetes, syphilis, etc. It has an acute and a chronic form.

A.—**Acute retrobulbar neuritis.**

SYMPTOMS.

1. *Headache*, in various degrees, sometimes quite severe.
2. *Orbital pain*, increased by movements of the eye and pressure upon it.

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[1] Read before the New York Academy of Medicine to introduce discussion of the subject, Dec. 18, 1890.

[2] On Neuro-Retinitis and Certain Cases of Fulminant Blindness. His *Archives*, xiv., 2, pp. 114–149 (see particularly pages 146–149), 1866.

[3] Amblyopia with Central Scotoma. *v. Graefe's Arch.*, xv., 3, p. 65, etc., 1869; and "Gräfe-Sämisch's Handbuch," vol. v., p. 809, 1876.

3. *Impairment of sight*, in all degrees. It may begin without any warning and advance rapidly, leading, in exceptional cases, to total blindness of both eyes in a day or two.

4. *Central scotoma*, for color and form. It may be of little intensity or so marked that no object is recognized in its area—partial and complete (or absolute) scotoma. The peripheric boundaries of the visual field remain normal.

5. *General diminution of color-perception.*

6. *Moderate retinal congestion and serous effusion* of the optic disc and adjacent retina. This condition may be followed by *ischæmia*, or the latter may be noticed from the beginning. The congestive swelling is to be considered as the distal boundary of the inflammatory focus, whereas the ischæmia is the result of the compression of the retinal vessels by the inflamed parts in the orbit.

7. The *termination* reveals three ophthalmoscopic pictures:

(*a*). Normal condition;

(*b*). Partial atrophy—always in the temporal half—of the optic disc;

(*c*). General atrophy of the optic nerves.

ETIOLOGY.

1. *Exposure and overwork.*

2. *Acute infectious diseases*, *e. g.*, measles, diphtheria, rheumatism, etc.

3. *Alcohol*, *nicotine*, *opium*, *lead poisoning*, etc.

4. *Suppression of menses.*

5. *No assignable cause.*

PROGNOSIS. Doubtful. We find complete and permanent recovery and partial recovery of sight, as well as permanent blindness, sometimes with preservation of one or several islets in the visual field, like oases in the desert. Their explanation later on.

TREATMENT. Rest, diaphoresis, salicylate of soda, mercury, iodide of potassium.

Let me detail one **illustrative case** from my own practice.

May 5, 1883, I was called to see Mr. Nathan Steinam, of New York City, forty years old, a successful merchant, healthy, without a constitutional taint, with no bad habits. The previous day he had complained of nausea and severe headache. In the evening his sight was dim ; the next morning he was totally blind in both eyes. I found S = 0, both pupils large, immovable, eyeballs of normal tension, their movements undisturbed. The media clear, the retinal arteries small, the veins enlarged and tortuous, the retina in the vicinity of the disc turbid, *i. e.*, a slight degree of congestive neuro-retinitis. The patient was a well man excepting his binocular blindness, and that he passed only a few ounces of urine during the day. The two next days the same condition. The urine drawn with a catheter still scant, but free from albumen, casts, and sugar. On the fourth day he passed more urine. Eyes the same, arteries more filled, but no pulsation on pressure ; veins dark, like thrombosed. Dr. Wiener, his family physician, Dr. Wm. H. Draper as consultant, and myself thought that the patient suffered from a uræmic attack. On the seventh day he had a spell of somnolence, but recovered soon and felt well. The excretions of urine became normal, the retinal arteries filled gradually to about two thirds of the natural calibre and pulsated on pressure, the veins lost their turgescence, the retina became transparent, and in about six weeks the optic discs were white, distinctly atrophic. Seven weeks after the onset of blindness he had, at various hours of the day, transient glimpses of light, during which he recognized the window and a lamp, both of which he correctly localized. When I came to see him I never could verify these observations, and was inclined to consider them as subjective sensations of light ; yet the patient, a man of well-balanced mind, positively asserted that he had seen light, though it disappeared quickly. He was treated with salicylate of soda, calomel *refracta dosi*, strychnia hypodermically, large doses of iodide of potassium, electricity. Dr. A. L. Loomis, in consultation, was of the opinion that some cerebro-spinal lesion would sooner or later manifest itself.

Thus far, almost eight years, no general disease whatsoever has appeared. The patient is physically and mentally as well as any person of his age. He goes regularly to his business, and is an intelligent and cheerful man, resigned to his loss of sight. But what has appeared is an islet of useful vision nasally and downward from the point of fixation in the visual field of the right eye, in which he counts fingers at a distance of 15′. Yet he takes in only three fingers at a glance and has slightly to move the eye to see the others. This represents an angle of a little over 1°. His optic discs are perfectly white, the blood-vessels about two thirds the normal calibre. The arteries pulsate on pressure. The picture resembles that of atrophy after total embolism of the central retinal artery. On examination, a few days ago, the patient's general and ocular conditions were found unchanged.

B.—**Chronic retrobulbar neuritis.**

SYMPTOMS.

1. *Diminution of sight*, binocular, gradual, yet in some cases with acute aggravations.

2. *Day blindness*, patients see better in the dusk, the disease first described by Arlt under the name of nyctalopia. The patients complain of a fog and are dazzled; at night they feel more comfortable, but in reality see no better.

3. *Diminished color-perception*, contrast shadows not distinguished.

4. *Central scotoma*, at first for colors, then for forms (absolute). Ring scotoma, by restitution of the central part.

5. *Diminished range of accommodation.*

6. *Boundaries of visual field normal.*

7. According to the stage of the disease we find *ophthalmoscopically:*

(*a*) Fairly normal condition or slight congestion.

(*b*) Sector-like atrophy in the lower outer part of the optic disc.

(*c*) General atrophy of the od.

OCCURRENCE.

Almost exclusively in males, and in the great majority of cases from abuse of *alcohol or tobacco*, or more frequently *of both*. Opium, stramonium, lead, carbon sulphide, malaria, syphilis, diabetes, rheumatism and gout are mentioned as causes. Of late cases have been reported as sequels of epidemic influenza.[1]

PROGNOSIS good in the first stage, when no atrophy of od.

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[1] Bergmeister, *Wien. klin. Wochenschr.*, 1890, No. 11. Landsberg, *Centralbl. f. Augenh.*, May, 1890, p. 141. Remak, *ibidem*, July, p. 201. C. M. Hansen, *West. Med. Reporter*, Oct., 1890, p. 231. Eperon, *Progrès Méd.*, Dec., 1890, p. 471.

is present. Rarely ending in complete blindness. The triangular atrophy of the od. may persist, but sight become normal again. Incomplete recovery is the rule.

TREATMENT.—Insist on total abstinence from alcohol and tobacco. Order strychnia, iodide of potassium, turkish baths, pure and invigorating air—sea, mountain, forest. Strychnia hypodermically, gr. $\frac{1}{30}$ or more, acts as an excitant and a tonic, with transient improvement of vision.

Diagnosis of cases of retrobulbar neuritis.

I beg to make a few remarks about the *examination of such patients.* The majority complain of impairment of sight. With test types at a distance we find both eyes affected, S from $\frac{20}{30}$ down to counting figures at several feet, excentrically.

Near by they require stronger glasses than their age and amblyopia would warrant.

Their field of vision has normal boundaries.

With the O S we notice one of the three stages: no abnormity, atrophy on temporal side, or general atrophy of the optic disc.

Examining for color-perception, we find that the patients of the last category are color-blind, those of the second recognize pigments well enough, but fail in contrast shadows; all of them show a color scotoma. The best mode to find this out is to hold a small piece of colored paper, not more than 3 *mm* long, with a pair of forceps in the visual line while you look at the patient's eye and let him look at yours. He will either be unable to see the color, especially green or red, or will confound it with another. If you want to measure the extent of the scotoma the handiest instrument is the perimeter of Schweigger. We shall, of course, not omit to examine the patient's general condition as to the well-known symptoms of alcoholism, and the other diseases mentioned before.

Pathological anatomy.

Von Graefe in 1866, and Leber in 1869, found the pallor in the temporal half of the od. and pronounced it to be the result of retrobulbar neuritis. Leber felt confident that the anatomical proof would soon be furnished. This was done in 1880 by Samelsohn[1] of Cologne, and Nettleship and W. Edmunds[2] of London. These investigators ascertained that in a limited area of the optic nerve there were augmentation of nuclei, hypertrophy of connective tissue, and waste of nerve fibres, *i.e.*, *interstitial, sclerosing inflammation*, in the same way as alcohol produces cirrhosis of the liver.

Following up the process from the eye toward the brain they discovered that the degenerated segment near the eye had the shape of a triangle, with its base on the lower outer margin of the nerve, its apex at the central vessels. Toward the brain it gradually passed from the periphery of the nerve to the centre, which it reached in the optic canal. A case of Vossius,[3] and one of Sachs,[4] confirmed these investigations. The authors followed the atrophic segment into the chiasm and the optic tracts.

The white sector in the od. seen with the O S is, of course, the direct observation, *intra vitam*, of the atrophic segment. I have repeatedly seen it, and pass a number of sketches around. By these investigations the course of the papillo-macular fibres through the optic nerve is traced, and we know now what is the lesion in central amblyopia from alcoholism and kindred affections. Some years ago, after my attention had been directed to this triangular optic atrophy in alcoholism, etc., I was astonished to find it clear and well marked in one eye of a healthy and absolutely temperate girl, of eighteen years of age. On further examination I noticed that there was, in that eye, **a coloboma of the macula lutea.** Since that time I have found the same in other cases of macular coloboma. This affection being either a congenital defect, or the result of choroidal hemorrhage or circumscribed exudation, gives, according to Gudden's law of ascending atrophy, a neat confirmation of the position of the macular fibres in the optic-nerve entrance. In a recent paper, by G. Lindsay Johnson,[5] of London, on extra-papillary colobomata, which is illustrated by excellently executed colored drawings, there are several cases of coloboma of the macula lutea which show this triangular atrophy of the od.

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[1] *Graefe's Arch.*, 1882, No. 1, pp. 1–109.

[2] Trans. Ophth. Soc. Un. Kingdom, 1881, p. 124, etc.

[3] *Graefe's Arch.*, xxviii., 3, p. 301.

[4] THESE ARCHIVES, 1889, p. 133.

[5] THESE ARCHIVES, 1890, pp. 1–29.

An excellent paper by Dr. W. Uthoff, of Berlin, appeared, four years ago, in *Graefe's Archiv*,[1] on the Influence of Chronic Alcoholism on the Human Visual Organ. Uthoff utilizes a clinical material of 30,000 patients (Schöler's Klinik), and has examined, in different asylums of Berlin, 1,000 inebriates functionally and ophthalmoscopically. He found the triangular white atrophy of the od. in 14%. Of six fatal cases he received the eyes and examined them with the microscope most thoroughly. I pass his plates round, exhibit some with the magic-lantern, and am happy to show you under the microscope some of his specimens which he had the kindness to present me with. The one shows the central interstitial neuritis in the period of nuclear infiltration, the other in the stage of connective-tissue hypertrophy. The process is characteristic by the limitation of its area of inflammation, circumscribed and triangular near the disk, of which we gain a view in the living with the ophthalmoscope, then oblong and round as it ascends toward the optic foramen. In one of the lantern specimens you see the changes pass like a band from the temporal border of the nerve straight across to the nasal border. This shows how the atrophy of the nerve may become total.

It is remarkable that in all the specimens a certain number of healthy nerve fibres are seen preserved in the atrophic parts. This explains on the one hand why alcoholists rarely become totally blind, and on the other why in the visual field of persons blind from retrobulbar neuritis islets of useful sight may permanently be preserved, oases in the desert, as I called them in the case reported before.

Retrobulbar optic neuritis presents an example of peripheric neuritis of great scientific and practical importance, the nature of which, correctly foretold by exact clinical analysis, has of late been verified in the most gratifying manner by pathologists. The process may start at different points of the nerve. Samelsohn found in his case the origin in the optic canal; in two of Uthoff's cases the changes in the nerve reached the optic foramen and entered the cranial cavity; in the four others they were present only in the distal end of the orbital portion of the nerve, at its entrance into the eye, and from 6–12 *mm* back. In all of these cases the temporal pallor of the od. was seen with the ophthalmoscope. In the fatal cases in which the ophthalmoscope showed no changes, none were found *post mortem.*

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[1] Year 1886, part iv., p. 95, and year 1887, part i., p. 257.

THE LENS-MEASURE OF THE GENEVA OPTICAL COMPANY.

By A. L. SMITH,
PRES. OF THE GENEVA OPTICAL COMPANY.[1]

(*With two drawings.*)

WE offer to the medical profession a new instrument intended to measure quickly and accurately the power of any form of lenses used in spectacles, and to give also the axis of cylinders. It is the invention of Mr. J. T. Brayton, of our company, and grows out of the necessity of having some means for readily determining the power of lenses without the very tedious method of neutralizing with the trial lenses. The essential features of the instrument are shown in two illustrations accompanying this communication. They are the natural size of the instrument. Of the three steel points (Fig. 1), and C B C (Fig. 2), the two outside ones are fixed, and the central, B, moves up and down easily and is held up by a spring. Upon pressing the face of a lens, as shown in the illustration, squarely and firmly upon the three points, the central point is depressed until all three touch the face of the lens, and the curvature of the lens determines the amount the central point is depressed. The lower end of the central steel rod impinges upon a segment of a cog-wheel, which mashes into a pinion carrying the index finger and controlling its movement, thus indi.

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[1] Having convinced myself by daily use of the practical value of the above instrument, I asked the Geneva Optical Company to kindly furnish for publication in these ARCHIVES a description, especially of the inner mechanism, of their handy apparatus, and also to mention the name of the inventor. I thank the President for his prompt and courteous compliance with my request. —EDITOR.

cating upon the dial plate the dioptries computed from the curvature of the face of the lens which is being tested. By rotating the lens upon the three points it is instantly seen if the side which is being tested is cylindrical, spherical, or plane. If a plane surface the index points to o (Fig. 1) in all meridians.

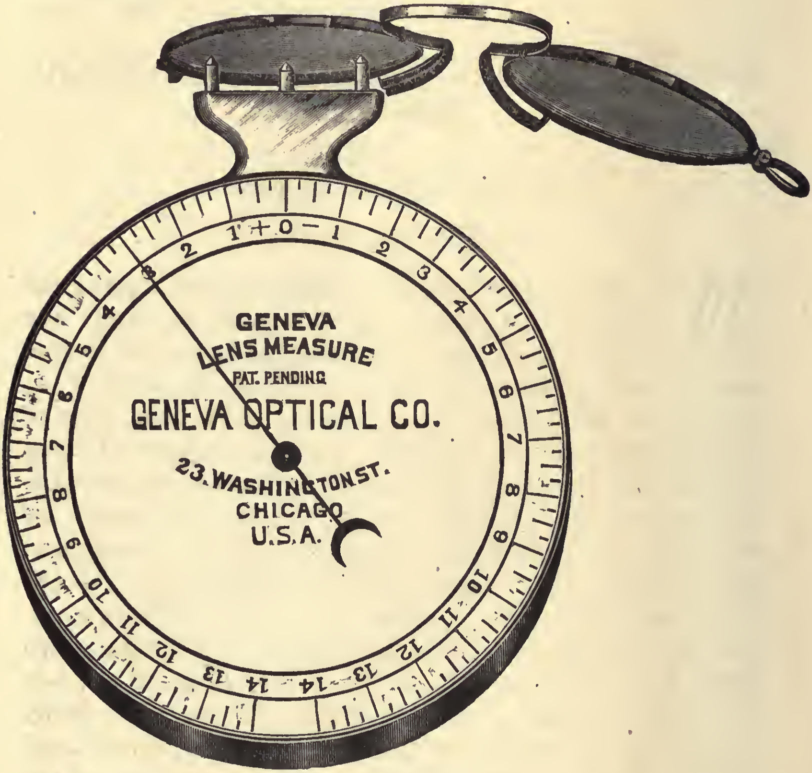

FIG. 1.

If a *cylindrical* surface it points to o when the three points are in line with the axis of the cylinder, and when rotated to the point where the highest degree of power is shown it indicates the refraction of that surface of the lens. By turning the lens over and testing the other side we instantly find the nature of that surface, and also read at once the power of a compound lens by using the two measurements given for each surface.

If it is a *bi-convex* or *bi-concave* lens we add the result of measuring each surface separately.

If a *periscopic* lens we deduct the less from the greater, for instance, if the concave side registers 1.25 and the convex plus 3.25 the lens is 2 dioptries.

The essential features of the instrument are the three points yielding to the curvature of the lens and connecting by suitable mechanism on the dial plate. The internal construction can be made in several methods on the system of levers, gears, and cogs, but the method used is the simplest form, using only a segment of a cog-wheel with teeth mashing into the central pinion (Fig. 2). There is a hair spring (G, Fig. 2) to take up the lost motion, the so-called "back-lash," of the centre pinion, also a heavy spring (H, Fig. 2) to force the central rod constantly upwards so that it stands normal above the two outside points as shown in the illus-

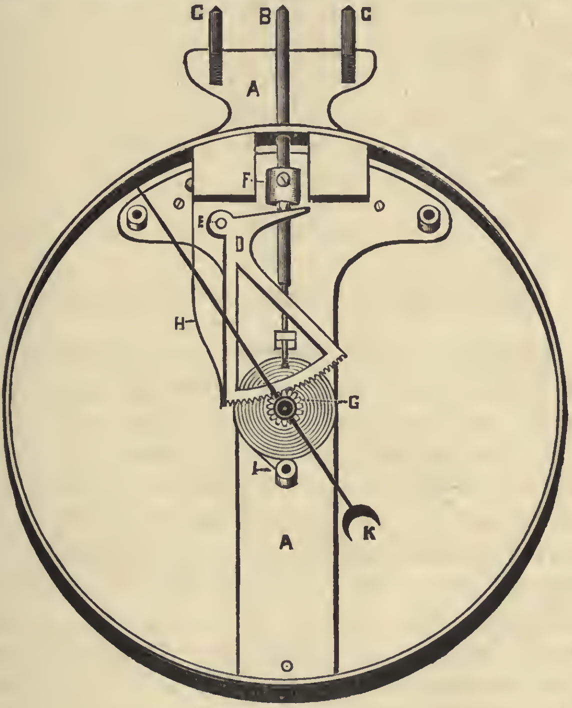

FIG. 2.

tration. "The two outside steel points are fixed, but can be moved if it is desirable at any time to adjust the instrument. The middle rod is attached to a solid bar (B, Fig. 2), which slides up and down in a slot in the ground plate (A, Fig. 2) of the instrument. The bar is attached to a life-edged chain in contact with the short arm of the rack D. The fixed point of the rack (or segment of the cog-wheel) (E, Fig. 2) is made adjustable by a screw device (F, Fig. 2). At the lower part of the rack are the cogs which work the pinion to which the pointer is attached" (Tilley).

As will be seen, the instrument analyzes a lens, measuring the curvature of each surface, as the grinder does by its gauges. From the curvature the refractive power is computed and expressed on the dial in dioptries, as indicated by the pointer. The refractive power of the lens is the combination of the power of both surfaces;—their sum, if both are either convex or concave; their difference, if the one is convex, the other concave.

SUPPLEMENTARY REMARKS BY H. KNAPP.

The instrument here described and illustrated (which can be easily taken apart and studied) is, as it were, a universal gauge of the surfaces of spectacle lenses, in which, for the convenience of the oculist, the refractive power corresponding to the curvatures is indicated on a dial by a pointer which is moved by a simple cog-wheel gearing. It was first exhibited and shortly described in the Ophthalmological Section of the American Medical Association, at its meeting at Nashville, in May, 1890, by Dr. Robert Tilley,[1] of Chicago. It should in medical literature be called the lens-measure or phacometer of I. T. Brayton. It is sure to become popular among oculists, for no one who has once seen it will be slow in adding it, as an almost indispensable supplement, to his trial case of lenses. It saves a great deal of time, and is as accurate as it is convenient.

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[1] *Journ. Am. Med. Assoc.*, Oct. 25, 1890, p. 597.

To test its accuracy I have tried eighteen of these instruments: sixteen were entirely exact, one like the other; one showed deviations here and there in the scale not exceeding $\frac{1}{8}$ dioptry—for instance, in a cylinder + 6 D the pointer marked the plane surface correctly 0, but the curved surface $5\frac{7}{8}$ instead of 6, as did all the other instruments; in one the hand of the dial pointed a little over $\frac{1}{4}$ D too much to the right all through. This can, of course, be easily corrected by removing the cover and setting the hand right. The inaccuracy of $\frac{1}{8}$ D in one or the other parts of the scale is probably owing to the incomplete elimination of the "back-lash" (*todter Gang*, in German, motion of machinery producing no effect), which, according to Nicholson, means the reaction upon each other of a pair of wheels, produced by irregularities of velocity, when the moving power is not uniform. The inaccuracy must be caused by some accidental derangement, for the instruments, being duplicates, should all be alike.

On the other hand, I have found far greater inaccuracies in my trial cases; for instance, in a new large case of P. Roulot's successor, $\frac{1}{4}$ D and more (up to 1 D in + 20 D) was not rare in the middle and higher numbers. As the Geneva Company's lens-measure will, without doubt, be generally introduced, it would be desirable to determine the shortcomings of the instrument or the inaccuracies in the numbering of our trial lenses, by means of a reliable phacometer—for instance, that of Snellen. The company say the computation of the refractive power from the curvature of lenses being based on the refractive index of crown-glass (to which that of pebble is practically equal), if a given lens has another focal distance than the one determined by their instrument, it is probably made of flint. In determining the axes of cylinders, I have found an inaccuracy (of about 5°) in only one of Roulot's case, which shows the remarkably exact work of this firm. The lens-measure, with printed instruction of its use, can be obtained from the company, or any reputable optician, at the price of $7,50 net.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FIRST HALF OF THE YEAR 1890.

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Sections I.–V. Reviewed by Prof. H. MAGNUS.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

a.—TEXT-BOOKS, MONOGRAPHS, TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS.

1. Deutschmann. Contributions to ophthalmology, first number. Hamburg and Leipzig, 1890.

2. Fox, Webster. A history of spectacles. *Med. and Surg. Rep.*, May, 1890.

3. Hirschberg. Egypt. Historical studies of an oculist. Leipzig, 1890.

4. Knies. Outlines of ophthalmology, with special consideration of the needs of students and physicians. Second edition, 1890. The first edition of this excellent book appeared less than two years ago. Thirty figures in the text. Price, $1.50.

5. Michel. Text-book of ophthalmology. Second revised edition. Wiesbaden, 1890. The first edition appeared in 1884.

6. Mooren. Visual disturbance and compensation. *Centralbl. f. allg. Gesundsheitspflege*, vol. ix., p. 217.

7. **Motais.** Hygiène de la vue dans les écoles et les collèges en France. *Comm. à l'acad. de méd.*, Nov. 19, 1890.

8. **Schlampp.** Guide to the clinical methods of examining the eye. 2d. ed. (In German.) Munich.

9. **Story.** The Irish licensing bodies and ophthalmic surgery. *Ophth. Rev.*, vol. ix., p. 37.

10. The hemianopic forms of the visual field and the centre of optical perception. An atlas of hemianopic defects. Wiesbaden, 1890. Excellent and exhaustive.

Deutschmann (1) presents some interesting clinical contributions, the special value of which lies in the thorough examination of the pathological anatomy of the cases. Papers on the following subjects are among the contributions: Injury to the eyes, and antiseptics. Remarks on the pathology of the optic nerve and its entrance. Homonymous hemianopsia after injury to the skull. On amaurosis and auto-intoxication in carcinomatosis. Spontaneous detachment of the retina with deep anterior chamber and marked decrease of tension. Glaucoma hemorrhagicum. Some rare forms of tumor of the eyes.

Fox (2), in his paper on the history of spectacles, introduces numerous illustrations, which are of great interest. He reviews modern history in particular, while the early history of glasses is embraced in a short review of well-known facts.

Hirschberg (3) has prepared three excellent historical papers. The second one is of special importance: "The ophthalmology of the ancient Egyptians," in which Hirschberg attempts to give an explanation of the eye diseases mentioned in the papyrus Ebers. This attempt is extremely difficult, and calls for extensive knowledge on widely different subjects; but in spite of the difficulties, Hirschberg has succeeded in a most admirable manner. The first paper: "Egypt as a climatic resort," is of general interest. In the third paper the examination of the first reports on Egyptian ophthalmia is notable.

Mooren (6) calls attention to the fact that the excellent method of Zehender of computing the loss of wage-earning power from visual disturbances is defective, because it takes into consideration only central vision. He proposes the universal acceptance of the formula computed by Jaesche on the basis of Zehender's formula.

11. Amsterdam. Seventeenth report for the year 1889.

12. Baltimore. Twelfth annual report of the Presbyterian Eye, Ear, and Throat Charity Hospital.

13. Herrnheiser. Report on the ophthalmic clinic of Prof. H. Sattler, for the year 1889. *Prager med. Wochenschr.*, 1890, Nos. 15 and 16.

14. Klein. Seventh annual report from the Ophthalmic Institute at Neisse. From February, 1889, to Jan. 31, 1890, inclusive.

15. Orlowsky. Report on the eye-patients in the private country practice for 1887 to 1889, inclusive. *Wjest. Ophth.*, May-June, 1890.

16. Rückert. Fourteenth report on the Ophthalmic Institute at Zittau for 1888 and 1889.

17. Schies-Gemuseus. Ophthalmic Institute at Basel. Twenty-sixth annual report from Jan. 1 to Dec. 31, 1889 inclusive.

18. Schreiber. Seventh annual report on the Ophthalmic Institute at Magdeburg from Jan. 1 to Dec. 31, 1889 inclusive.

19. Stuffler. Relazione clinico-statistica del quadrimestre Luglio–Ottobre, 1889. *Rass. di scienz. med.*, vol. v.

20. Utrecht. Thirty-first annual report.

21. Wagner. Short report on the City Ophthalmic Institute at Odessa for 1888 and 1889. *Wjestn. Ophth.*, May–June, 1890.

22. The Wills Eye Hospital. Report for the year ended Dec. 31, 1889. Philadelphia, 1890.

23. Wolffberg. On statistics of eye-diseases, with a diagram for affections of the lids. Supplement to the jubilee report in honor of the twenty-fifth anniversary of the existence of the Institute. Breslau, 1890.

Gunning (11) reports on 8,128 patients, with 41 extractions and 116 tenotomies.

In Baltimore (12) 5,924 patients were treated ; 33 extractions with, 60 without, iridectomy.

In Prague (13) 3,430 new patients were treated ; May and June furnished the largest number. Operations, 1,026, among them 213 extractions according to Graefe, only 1.5 % of which failed on account of suppuration. Iridectomies, 158. In one case a cysticercus was extracted from the vitreous.

Klein (14) treated 1,282 new patients and performed 146 operations, among them 28 extractions.

The number of eye-patients (15, Orlowsky) was 2,039, being equal to 19 % of the total number, 10,686. Detailed figures cannot be given. In trachoma, which comprised 20 % of all eye-patients, ORLOWSKY carefully presses out the granulations with the fingers, repeatedly, if necessary, washes out the conjunctival sac with a solution of sublimate, 1:1,500, and rubs iodoform powder into the bleeding parts. The following day he touches the lids with a solution of nitrate of silver (2 %), or of sulphate of copper in glycerine (6 %). When the secretion ceases and no new granulations appear, the patients are discharged with the yellow salve (1:120). He claims that the granular form is cured by this method in from four to six weeks. In the treatment of blennorrhœa and hypopyon iodoform and sublimate solutions are extensively used. The number of major operations was 379 on 260 patients, among them 220 extractions, with a loss of 3.2 %. HIRSCHMANN.

In ZITTAU (16) 6,246 new patients were admitted in the last two years. Number of extractions, 129. An obituary of Just is added to the report.

At BASEL (17) 2,206 patients were treated at the dispensary, 531 clinically. A large number of clinical histories are added.

SCHREIBER (18) treated 2,046 patients. Twenty extractions according to Graefe, with one loss. Number of extirpations of the lachrymal sac, 36. He adds a short paper on bromethyl narcosis and a clinical history (primary sarcoma of the ciliary body).

In the ophthalmic division, in charge of Saltini, of the hospital at Modena 1,268 eye-patients were treated during the four summer months according to STUFFLER (19). Number of operations, 118. The writer advocates the use of a 3-% solution of nitrate of silver in trachoma. DANTONE.

In UTRECHT (20) 3,392 new patients were treated. Number of extractions, 47.

The number of patients (21, Odessa) for 1888 was 2,862, in-door patients, 462 ; for 1889, 3,280, in-door patients, 423. Number of extractions according to Graefe, in 1888, 122 ; all good ; in 1889, 106, with 5 losses. HIRSCHMANN.

In PHILADELPHIA (22) 9,436 new patients were treated. Extractions, 86 with, 5 without, iridectomy ; 63 enucleations.

WOLFFBERG (23) justly calls attention to the numerous sources of error which the statistics of the different institutions contain, and makes propositions to overcome them.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

a.—GENERAL PATHOLOGY AND DIAGNOSIS.

24. Basevi. Delle imagini suggettive. *La Spallanzani*, vol. xxviii., 5.

25. Garnier. The changes in the ciliary muscle under the influence of age and refraction. *Wjestnik Ophth.*, Mar.–Apr., 1890.

26. Naumow. On some pathologico-anatomical changes in the fundus of the new-born child. *Inaug. Diss.*, St. Petersburg, 1890.

In 11 cases of chronic glaucoma and 14 of idiopathic affection of the optic nerve, Basevi (24) has examined the development, nature, and fading of the after-images, resulting from the influence of the individual, monochromatic spectral colors. The differences between the two diseases named, and between them and the healthy eye, were so marked that the author thinks they could be used for diagnostic purposes. Dantone.

From microscopic examination and measurements of the ciliary muscle in eyes of persons of different age and refraction, Garnier (25) arrived at the following results : in new-born and prematurely-born children the part of the ciliary muscle adjacent to the insertion of the iris is drawn forward with the root of the iris, there being no anterior chamber. The anterior chamber is not the result of the functioning of the eye, but of progressive development, as he discovered in an eye of a child born six weeks beyond the time. The circular fibres are present in the ciliary muscle of all new-born children, and must be considered an integral part of that muscle. Exercise, of course, can develop the muscle (functional hypertrophy) ; the circular fibres may increase, which, however, depends less upon the original refractive condition of the eye (which is usually hypermetropic in the new-born) than upon the demands made upon it. The structure and form of the ciliary muscle vary very considerably. The author doubts a separate development, or atrophy of particular parts of the muscle, on account of the uniform action and innervation of its different parts. Hirschmann.

Naumow (26) examined under the microscope 47 pair of eyes of mature children which had died during birth, or soon afterwards, and 22 pair of eyes of prematurely born children (10 pair of 7 months' and 12 pair of 8 months' children). Nothing pathological was found in the latter. In 12 pair—*i.e.*, 25.5 per cent. of the mature children, there were pathological changes as follows: hemorrhages in and upon the retina and choroid, œdema of different parts of the retina and of the optic nerve, infiltration, with a small-celled mass and optic neuritis in an early stage. The hemorrhages are particularly numerous in the region of the macula, and are sometimes quite large; they generally occur in the inner granular layer, seldom in the layer of ganglion cells; in the peripheric parts of the retina, however, they generally occur in the latter layer, rarely in the former. Sometimes they are small, but occasionally rupture the membrana limitans interna. Naumow attributes congenital weakness of sight to such hemorrhages in the region of the macula lutea. Congenital amblyopia and atrophy of the optic nerve may result from the choked disk mentioned above. The so-called central coloboma he ascribes to a large hemorrhage which had occurred during birth. HIRSCHMANN.

b.—GENERAL THERAPEUTICS.

27. COSTOMIRIS. Du massage oculaire au point de vue historique et therapeutique et surtout du massage direct de la conjonctive et de la cornée. *Arch. d'ophth.*, vol. x., No. 1.

28. MEIFFERT. Considérations sur l'énucléation de l'œil. *Thèse de Lyon*, 1889.

29. SCHEFFELS. On resection of the optic nerve. *Zeh. klin. Monatsbl. f. Augenhk.*, 1890, p. 197.

30. SIMI. Sulla enucleazione. *Boll. d'ocul.*, vol. xii., p. 3.

COSTOMIRIS (27) begins his somewhat lengthy paper with an historical review, which shows that massage of the eye was known to the old Greeks, and was used by Hippocrates and Galenus according to definite rules. A distinction was made according to quantity and quality, just as the author to-day uses strong, moderate, or gentle friction, which again may be brief, moderately long, or prolonged. Direct massage was not known to the ancients. This form in particular is preferred by him, and he contends that the proper form of massage, adopted to each individual case, is capable of curing even the most inveterate affections of the conjunctiva and cornea. He then describes in detail the different kinds of massage and their indications. He thinks that the remedial action must be ascribed to the massage alone, though he uses different remedies, and recommends for strong friction pulverized boracic acid, while for moderate and prolonged friction a salve of boracic acid, or of boracic acid with acetate of lead, or of white precipitate, is useful. The yellow and iodoform salve frequently cause irritation, and are suitable only for obstinate cases. After massage the eyes are washed with collyria of acetate of lead, nitrate of silver, or boracic acid, etc. Massage can be extensively employed in trachoma with good results, as the author shows from different cases. He has also used massage with good effect in acute affections of the conjunctiva, even the severest, like blennorrhœa, croup, and diphtheria ; also in scleritis, episcleritis keratitis parenchymatosa, hypopyon, in infectious corneal ulcers, affections of the lids, œdema, echymoma, and tenonitis. In internal affections massage is useless, except when, as in secondary glaucoma, iritis, or cyclitis, it is due to a corneal affection. v. MITTELSTÄDT.

From the experience of Gayet, MEIFFERT (28) prefers enucleation to every other operation, for instance, evisceration, amputation of the anterior portion, etc. Recovery is rapid, and of course there is no chance for vision in any of the other operations. A conjunctival suture after enucleation is rather injurious than useful. MARCKWORT.

SIMI (30) thinks that enucleation of the eyeball has been abused. No general indications can be formulated, but every case must be carefully considered. DANTONE.

III.—INSTRUMENTS AND REMEDIES.

a.—INSTRUMENTS.

31. CHIBRET. Un optomètre de poche fondé sur une propriété des progressions. *Soc. d'ophth. de Paris*, Feb. 4, 1890.

32. LEPLAT. Un instrument pour controler l'orientation des verres cylindriques. *Arch. d'ophth.*,vol. x., 1, 1890 ; *Soc. d'ophth. de Paris*, Feb. 4, 1890.

33. MADDOX. A new test for heterophoria. *Ophth. Rev.*, vol. ix., p. 129.

34. HARLAN. Periscopic cylindrical and sphero-cylindrical glasses. *Trans. Amer. Ophth. Soc.*, 1889.

LEPLAT (32) has constructed an instrument for testing the cylindrical glasses furnished by the optician, especially the position of the axes. A ring divided into degrees and provided with a handle, is fitted with the glasses necessary for neutralizing ; the glass to be tested is placed on a second ring concentrically fitted into the first and provided with an indicator. The author then describes the simplest methods for testing the different simple and combined cylindrical glasses. v. MITTELSTÄDT.

b.—REMEDIES.

35. GRÄFE–JÄNICKE. A contribution to the knowledge of pyoktanin. *Fortschr. d. Med.*, 1890, Nos. 11 and 12.

36. PANAS. Sur l'action anesthésique locale de la strophantine et de l' ouabaïne. *Arch. d'ophth.*, 1890, No. 2, Mar.–Apr.

In GRAEFE'S (35) clinic Stilling's claims of the remarkable value of the aniline dyes as antiseptics were subjected to a thorough test, the result of which was very unfavorable. Graefe's results, like those of most other authors, were unsatisfactory. As regards their clinical value, Graefe scarcely ever obtained any real results ; in one case possibly a good result was observed, as he succeeded in preventing a threatening panophthalmitis in a case of perforating corneal injury. In the great majority of cases treated with pyoktanin no effect was observed, while not a small number were directly injured by it. Careful bacteriological tests showed that the germ-destroying power of the aniline dyes was much less in blood-serum than in distilled water. The various kinds of bacteria reacted differently, and were checked in their development by various solutions. Staphylococci were killed by a solution of 1:1,000 to 5,000 in half a minute, but by a solution of 1:20,000 only after ten minutes. A severe inflammation ensued in rabbits' eyes when they were touched with a pyoktanin pencil 1:1,000 ; the local irritation does not seem to be as slight as Stilling claims.

PANAS (36) reports on his experiments of anæsthesia of the eye with strophantine and uabaïne. In rabbits the anæsthetic effect appeared later than after cocaine, and lasted up to three hours. There were no symptoms of irritation, and the pupil remained unchanged except a slight dilatation after strophantine. In the human eye uabaïne caused neither irritation nor loss of sensibility, while strophantine rendered the cornea insensible after fifteen minutes, the loss of sensibility lasting two hours ; it cannot be used, however, on account of the severe reaction of the conjunctiva and the marked painful smarting. An inflamed eye is not affected by strophantine. v. MITTELSTÄDT.

IV.—ANATOMY.

37. BECHTEREW. On the visual area at the surface of the cerebral hemispheres. *Arch. f. Psychiatr.*, 1890, No. 1.

38. DARKEWITSCH. On the decussation of the optic-nerve fibres. *Wratsch*, 1890, Nos. 5 and 6.

39. EWART. On the development of the ciliary or motor-oculi ganglion. *Royal Soc. London*, March, 1890.

40. MUNK. Visual area and the movements of the eyes. *Sitzungsber. d. preuss. Acad. d. Wissensch.*, 1890, iii.

41. PFISTER. On the form and size of the intervaginal space of the optic nerve within the optic canal. *Arch. f. Ophth.*, vol. xxxvi., 1.

BECHTEREW (37) arrived at the following results: (1) By unilateral destruction of the cortex in animals (dogs and cats) three kinds of visual disturbance can be produced: (*a*) binocular homonymous hemianopsia; (*b*) binocular homonymous hemianopsia with incomplete general blindness (amblyopia) of the eye of the opposite side, especially impairment of its part of most distant vision; and (*c*) incomplete blindness of the opposite eye. (2) The region of the cortex, the destruction of which causes visual disturbances, extends from behind forward, over the whole occipital and a large part of the parietal lobe, and laterally from the temporal to the inner edge of the occipital lobe, spreading out upon the inner surface of the latter. (3) In the cortex of the hemispheres there exist in reality two visual areas, which to a great extent overlap each other: the one, embracing the occipital and parietal lobes, controls the function of the corresponding halves of the retinæ; the other, lying in its greatest extent in the region of the parietal and only partly in the occipital lobe, influences the function of the opposite part of the retina. (4) Destruction of the first-named area produces homonymous hemianopsia of both eyes; destruction of the other is generally only possible with that of the first also, producing in the animal homonymous binocular hemianopsia with amblyopia of the opposite eye, especially obscuration of the region of most distinct vision. (5) The crossed amblyopia sometimes observed must be ascribed to an affection of the second area. (6) In homonymous hemianopsia due to destruction of the cortex the region of most distinct vision remains unaffected. (7) The hemianopsia due to destruction of the occipital lobe is either a complete and apparently permanent one, or incomplete (hemiamblyopia) and temporary, according to the extent of the destruction. (8) After the re-establishment of sight in limited destruction of the occipital lobe a second complementary destruction again produces hemianopsia of shorter or longer duration. (9) In cats and dogs it is never possible by destruction of the cortex to produce a visual disturbance of the eye of the same side alone or limited disturbances of the eye of the opposite side in form of the blind spot of Munk. (10) Extensive destruction on both sides in the region of the parietal and occipital lobes produces complete blindness of longer or shorter duration. (11) Binocular (homonymous) hemianopsia with incomplete blindness of the opposite eye in some cases passes in the course of time into pure homonymous hemianopsia, in others into crossed incomplete blindness. (12) The visual disturbance resulting from destruction of the visual area of animals, and considered mental blindness by some authors, is nothing but an impairment of vision of an amblyopic nature. HIRSCHMANN.

Michel's last publication (on degeneration of the optic nerve and decussation of the optic-nerve fibres) induced DARKEWITSCH (38) to examine similar specimens in his collection and make new experiments, from which he concludes that "Michel's latest investigations by no means disprove the old assertion of Gudden, that in the higher animals an incomplete decussation takes place." Darkewitsch partly ascribes the opposite results of Michel to the fact that he has mistaken in his specimens the fibres of Forel's decussation for fibres of Meynert's commissure and the latter for fibres of Gudden's commissure. For the detailed proofs of this see the original. The author relies for proof mainly on the division in new-born cats (through the parietal bone) of the optic tract at the point where it passes upon the corpus geniculatum externum. Examination of the animal killed forty-two days after the operation (total division of the left tract) showed : left thalamus opticus hardly half as large as right ; the two corpora geniculata atrophied on the right side ; the left optic tract has entirely disappeared (in its place only Meynert's commissure is visible) ; the right tract is present, but thinner (on account of the atrophy of Gudden's commissure), than in an animal not operated upon ; both optic nerves are present, the right thinner than the left, but both thinner than in an animal not operated upon. Both optic nerves contained uninjured fibres.

HIRSCHMANN.

Munk (40) assumes that the focussing of the eyes is caused by sensations of light, from which the excitation extends in the radial fibres of the corona radiata directly to the subcortical parts of the brain. These fibres serve the involuntary movements of the eyes, which permit the latter to wander and fix themselves upon what had previously been seen indistinctly; these movements must be considered a special kind of congenital reflex, the lowest visual reflexes, which do not presuppose conscious visual impressions, but only sensations of light.

Pfister (41) found that in very many cases there is a communication between the subdural space of the brain and the intervaginal space of the intra-orbital part of the optic nerve, passing completely around the latter. A distinct arachnoid sheath separate from the pia and dura mater does not seem to exist in the optic canal. The dura mater, however, is markedly developed, especially below the optic nerve, where the superior ophthalmic artery lies.

V.—PHYSIOLOGY.

a.—RETINA AND GENERAL PHYSIOLOGY.

42. Basevi. Influenza dell' adattamento sulla sensibilità retinica per la luce e per i colori. *Ann. di ottalm.*, vol. xviii., 6, p. 475.

43. Berry. A suggestion as to the function of some of the retinal elements. *Ophth. Rev.*, vol. ix., p. 134.

44. Charpentier. Recherches sur la persistence des impressions rétiniennes et sur les excitations lumineuses de courte durée. *Arch. d'opht.*, vol. x., 2 and 3.

45. Laqueur. On pseudentoptic visual perceptions. *Arch. f. Ophth.*, vol. xxxvi., 1.

46. Lawrentjew. On the influence of drawing, printing, and female fancy work upon sight. *Wjestnik obsch. Hygiea sudjebn. i pact. med.*, vol. vi., 1; *Wratsch*, 1890, No. 21.

47. Mendelsohn. Investigations on the differential sensibility of the visual organ. *Minutes of the viii. Meeting of Russian Physicians and Naturalists.*

48. Sachs. The causes of the apparently greater proximity of the lower of two double images placed above each other. *Arch. f. Ophth.*, vol. xxxvi., 1.

49. SCHUBERT. The position of the book and the direction of the writing. Hamburg and Leipzig, 1890.

BASEVI (42) has made extensive investigations on himself and nine other young persons with normal sight as to the adaptability of the retina for white and colored light, and on the whole has obtained about the same results as Aubert, Peschel, and Treitel. The eyes were put into a condition of rest by a stay, for 15 to 20 minutes, in a room darkened with heavy curtains and therefore not absolutely dark. The author found that after adaptation sight had been increased threefold, measured with Snellen's test-types, while the sense of light, measured with Treitel's tests, had been increased eighteen-fold. The latter experiment was made at a greater angle than that used by Aubert, which accounts for the great difference of result. While measuring himself with a Foerster photometer the author, while in the state of adaptation, was able to recognize the same lines as before, though the illumination had been reduced to $\frac{1}{160}$. After the eyes had been rested and light was gradually admitted, the colors were recognized in the following order: red, yellow, green, and finally blue. When the eye was exhausted for one color by a corresponding glass, that color took the last place in the row. The author found the visual field enlarged, also that for colors; but the period of rest must be longer and the test must take place in greater darkness. On the basis of these observations the author concludes that hemeralopia is a disturbance of the adaptability of the eye.

DANTONE.

CHARPENTIER (44) tries to determine the influence which the intensity, duration, and color of a luminous excitation falling upon the retina exert upon the duration of the sensation apparently continuing with the same intensity after the excitation has ceased. (For description of the apparatus and the manner of experiment see the original.) He first decides the question as to the relation to each other of the duration and intensity of a luminous excitation in their effect upon the retina. He found, as others did before him, that within a space of $\frac{2}{1000}$–$\frac{125}{1000}$ of a second the amount of light necessary for perception is inversely proportional to the time of its action, and that doubling of a certain amount of light, the time of its action remaining the same, is equivalent to doubling the duration, the intensity remaining the same. If we imagine an excitation subdivided into a large number of smallest elementary irritations (impressions élémentaires), these elementary irritations unite within a certain time as the sensation of light increases.

Sachs (48) explains the apparently greater proximity of the lower double image in the first place by its being referred to the horopter, and in the second place by the increase in accommodation when the eyes are directed downward.

b.—COLOR-PERCEPTION.

50. Bickerston. Color-blindness. *Brit. Med. Jour.*, March, 1890.

51. Edridge-Green. Two new tests for color-blindness. *Brit. Med. Jour.*, Jan., 1890.

52. Fick. The theory of color-sense in eccentric vision. *Arch. f. d. ges. Physiol.*, vol. xlvii.

53. Grossmann. Note on tests for color-blindness. *Brit. Med. Jour.*, Jan., 1890.

54. Hering. The diagnosis of color-blindness. *Arch. f. Ophth.*, vol. xxxvi., 1.

55. Hess. On the changes in the spectral colors produced by exhaustion of the retina with homogeneous light. *Arch. f. Ophth.*, vol. xxxvi., 1.

56. Hogg. An undetected case of color-blindness. *Brit. Med. Jour.*, April, 1890.

57. Raymond. Une observation colorée. *Gaz. des hôp.*, 1889, p. 680.

58. Uhthoff. Further investigations on the dependence of the acuteness of sight upon the intensity, as well as upon the wave-length in the spectrum. *Arch. f. Ophth.*, vol. xxxvi., 1.

Fick (52) defends the theory of three colors against the attacks of Herring and Hess, and shows that one of the primary colors (blue) has been undoubtedly shown to exist.

Sections VI.–XI. Reviewed by Prof. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

59. Fukala. Operative treatment of high degrees of myopia by aphakia. *Arch. f. Ophth.*, vol. xxxvi., 2, p. 230.

60. Hirschberg, J. Diabetic myopia. *Centralbl. f. prakt. Augenhk.*, 1890, p. 7.

Myopia, hypermetropia, and astigmatism are the subjects of sixteen other papers, which offer nothing essentially new, and are therefore omitted in this report.—K.

FUKALA (59) has performed discission on nineteen myopic eyes with a myopia from 13 D upward. They were all young persons under twenty-four, with relatively good sight. As a result of the aphakia such myopes see better at a distance, the retinal images are enlarged, the excessive effort at convergence and its injurious consequences are removed, binocular vision for near objects is re-established, and the spasm of accommodation and the accommodation itself—two factors injuriously affecting highly myopic eyes—are done away with.

HIRSCHBERG (60) describes three cases in which a low degree of myopia had been developed in eyes good until then, in consequence of diabetes.

VII.—LIDS.

61. GRADL. The treatment of blepharitis squamosa. *Centralbl. f. prakt. Augenhk.*, 1890, p. 112.

62. CIRINCIONE. Cisto-adenoma sottocutaneo giustaposto al sacco lagrimale. *Progresso med.*, vol. iv., 18.

63. FRICKE. Case of congenital defect of the lid. *Zeh. klin. Monatsbl.*, vol. xxviii., p. 53.

64. FUCHS, E. On isolated bilateral ptosis. *Arch. f. Ophth.*, vol. xxxvi., 1, p. 234.

65. GASPARINI. Innesto di cute in caso di simblefaro con buon esito definitivo. *Ann. di ottalm.*, vol. xix., 1, p. 89.

66. LANDOLT, E. Un nouveau procédé d'opération dans le distichiasis. *Arch. d'opht.*, vol. x., 1, p. 1.

67. NICATI. Nouveau procédé de marginoplastie palpébrale. *Arch. d'opht.*, vol. x., 2, p. 160.

68. PINCUS, O. Remarks on Jacobson's operation for trichiasis. *Centralbl. f. prakt. Augenhk.*, 1890, p. 259.

69. ZIRM. The application of oblique blepharotomy. *Wiener klin. Wochenschr.*, 1890, No. 14.

GRADL (61) has treated blepharitis squamosa with a 5-% aqueous solution of chloral hydrate alternately with a pyrogallol salve (1:8). A 2-3-% sulphur ointment acted still more favorably.

CIRINCIONE (62) observed in a woman aged fifty a small tumor about the size of a bean, which lay between the cutis and the outer wall of the lachrymal sac, was slightly movable, but otherwise closely resembled an ordinary tumor of the lachrymal sac. After the death of the patient the tumor was examined, when it proved to be a free cyst, which sprang neither from the cutis nor from the wall of the lachrymal sac, with thickened walls and transparent yellowish contents. After a careful microscopic examination, the author declared it to be an imperfectly developed adenoid, which had originated from the cellular elements of the mucous glands of the lachrymal sac. DANTONE.

In a girl aged seventeen, FRICKE (63) saw in the right eye a large congenital coloboma of the upper lid and an indicated one of the lower. In the left eye there was a small coloboma of the upper lid and a dermoid tumor at the corneal margin. In both there was at the outer canthus a lipoma resembling a dermoid.

In connection with the description of five cases of isolated bilateral ptosis in otherwise healthy women, which in three had developed at a more advanced age, and in two had existed since childhood, FUCHS (64) expresses the belief that this affection must be ascribed to atrophy of the levator. The course of the disease is always a very slow one, so that even after the lapse of many years a slight contractility of the muscle can be demonstrated. There is also atrophy of the soft parts over the levator; in some cases a slight degree of ptosis has gone before, indicating an abnormal condition of the levator. This kind of atrophy cannot be classified under any of the forms of muscular atrophy thus far known; it is, therefore, a special variety of atrophy which is confined to the levatores palpebrarum.

GASPARINI (65) reports another case of successful transplantation of skin, the transplanted piece being placed upon the raw surface after a symblepharon operation. The edges of the graft, which was taken from the upper arm, changed to mucous membrane, while the remainder preserved the nature of epidermis, and therefore had a pale-gray color. The impaired mobility of the two eyes operated upon was removed, and the successful result had remained six months after the operation. DANTONE.

For the removal of total distichiasis, LANDOLT (66) recommends the following method: The whole upper lid is divided into two layers: the anterior being composed of the skin, subdermal tissue, and eyelashes; the posterior, of the muscle, tarsus, and conjunctiva. An incision is then made a few millimetres from the margin along the whole length of the anterior layer, dividing it into a smaller lower and a larger upper section. The lower one, being thus converted into a bridge of skin, is then displaced upward as far as possible beneath the upper, with the aid of two threads, each armed with two needles, passing them beneath the orbital margin and tying them there; the lower edge of the upper flap thus comes in contact with the edge of the layer composed of muscle, tarsus, and conjunctiva, and unites with it. The narrow bridge of skin, from which the eyelashes have previously been removed (though leaving them does not retard the healing), becomes attached beneath the flap covering it, though an incision through the latter parallel to the edge of the lid must be made a few days afterward, to allow the eyelashes to pass through, which have then attained their permanent position.

In operating for trichiasis resulting from atrophy of the edge of the lid by means of marginal transplantation, NICATI (67) does not take the flap from the affected lid, as he formerly did, but from the opposite one. His method is as follows: (1) Incision into the margin of the lid parallel to it, so as to gain space for the transplantation. (2) Formation of a flap 1-2 *mm* wide extending the whole length of the other lid and attached to its base. (3) Insertion in the groove of the affected lid, a loop of thread being passed horizontally through the base of the flap and through the ends of the groove, and then tied. (4) Three days later division of the base of the flap and separation of the lids.

Doubts have lately been expressed as to the merits of Jacobson's operation for trichiasis, transplantation of a pedunculated flap into the intermarginal space. PINCUS (68) thinks that these objections are unfounded, provided the transplanted piece of skin is entirely devoid of even the smallest hairs, and it is not made too broad. (The implantation of narrow flaps without pedicles, according to W. F. Smith, gives very good results.—H. K.)

ZIRM (69) recommends oblique blepharotomy for the cure of spastic entropium, senile entropium, and entropium under the bandage. The operation consists in dividing one of the eyelids in its entire thickness by a single oblique incision from the outer canthus.

VIII.—LACHRYMAL APPARATUS.

70. Berlin, R. Extirpation of the lachrymal sac. *Corresp. Bl. d. allg. Mecklenb. Aerzte-Vereins*, 1890, No. 126, p. 282.

71. Chibret. Ablation de la grande lacrymale palpébrale. *Soc. d'opht. de Paris*, Feb. 4, 1890.

72. Gillet de Grandmont. Valeur de l'élongation des points lacrymaux. *Soc. franç. d'opht.*, May 5, 1890.

73. Hirschberg, J. Mumps of the lachrymal glands. *Centralbl. f. prakt. Augenhk.*, 1890. p. 77.

74. Kieselstab. On epiphora. *Münchener med. Wochenschr.*, 1890, No. 34.

75. Nicati. Drainage du sac lacrymal. *Arch. d'opht.*, vol. x., 2, p. 160.

76. Scheffels, O. A case of acute bilateral, non-purulent inflammation of the lachrymal gland. *Centralbl. f. prakt. Augenhk.*, 1890, p. 136.

77. Seggel. Occlusion of the lachrymal points by contraction of the sphincter. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 362.

78. Venneman. Traitement de la fistule lacrymale. *Soc. franç. d'opht.*, May 5, 1890.

79. Webster, D. Acute dacryocystitis. *N. Y. Med. Journ.*, vol. li., No. 12.

Chibret (71) removed the palpebral lachrymal gland in twenty-eight cases, and thinks he can recommend this treatment. Masselon is of the same opinion, while Galezowski, Gorecki, and Meyer are more or less opposed to it. Meyer knows of a case of atrophy of the optic nerve in consequence of suppuration following removal of the palpebral lachrymal gland.

Marckwort.

Gillet de Grandmont (72) does not divide the lachrymal points, but dilates them with a conical probe and sounds. In suppuration of the lachrymal sac he washes it out with antiseptics. In occlusion of the lower opening of the lachrymal canal by the hyperæmic mucous membrane of the inferior turbinated bone, he treats the latter with the galvano-cautery or removes a piece of it; Armaignac particularly avoids every "mutilation" of the lachrymal apparatus; Galezowski and Abadie are adherents of the usual treatment with Bowman's probes. When this method fails, Abadie removes a part of the lachrymal gland; Swartz de Mendoza insists that the canaliculus should not be split more than 1½–2 *mm;* Terson is not satisfied with any of the present methods.

MARCKWORT.

HIRSCHBERG (73) considers mumps of the lachrymal gland a sub-acute, non-purulent, bilateral inflammation, which causes a disfigurement of the face. He describes two such cases, which healed under lukewarm applications and iodide of potassium.

KIESELSTAB (74) calls attention to the fact that epiphora may depend upon the condition of the turbinated bones.

In suppuration of the lachrymal sac, especially the chronic form with dilatation, NICATI (75) makes an extensive incision with a very broad knife from the slit canaliculus, inserts at once a probe 3 *mm* thick, and afterwards a lead wire made of several strands, twisted together, which remains in place for three days. In almost every case this is sufficient to effect a cure. The epiphora, which sometimes lasts for a while, is due to granulations, and soon ceases. If the sac should become distended again and the introduction of probes is not followed by rapid improvement, the procedure must be repeated. v. MITTELSTÄDT.

SCHEFFELS (76) reports a case of bilateral acute inflammation of the lachrymal gland, which soon passed away after the use of iodide of potassium and applications of acetate of lead. He considers it of an infectious nature.

In lachrymal fistulas VENNEMAN (78) recommends the introduction of a strip of gauze soaked in lactic acid, in order to produce closure of the fistula. The strip is introduced once a day, and an antiseptic bandage placed over it. He claims that under this treatment the fistula closes in one to six weeks.

MARCKWORT.

IX.—MUSCLES AND NERVES.

80. AEBERLI, H. Contributions to the knowledge of nuclear paralysis of the ocular muscles. *Inaug. Diss.*, Zurich, 1890.

81. BASEVI. Le quadrigemelle come centri di azione reflessa sui movimenti oculari. *Ann. di ottalm.*, vol. xviii., 6, p. 520.

82. DUFOUR, A. Les paralysies nucléarires des muscles des yeux. *Ann. d'ocul.*, vol. ciii., p. 97.

83. GRANDCLÉMENT. Paralysie essentielle et temporaire de convergence chez les adolescents. *Soc. franç. d'opht.*, May 5, 1890.

84. JAVAL. De la stéréoscopie dans le traitement du strabisme. *Soc. f. d'opht. de Paris*, Feb. 4, 1890.

85. Lawford, T. Paralysis of ocular muscles in congenital syphilis. *Ophth. Rev.*, 1890, p. 97.

86. Mangnos et Grasset. Un cas de paralysie alterne de l'oculomoteur avec aphasie traumatique. *Progr. méd.*, 1890. No. 6, p. 105.

87. Meyer, A. On a case of ophthalmoplegia bilateralis perfecta in tabes dorsalis. *Inaug. Diss.*, Berlin, 1890.

88. Standish, Myles. Partial tenotomies in cases of neurasthenia with insufficiencies of the ocular muscles. *Trans. Amer. Ophth. Soc.*, 1889.

89. Wadsworth, O. F. Spastic torticollis, apparently due to faulty position of the eyes and cured by tenotomy. *Trans. Amer. Ophth. Soc.*, 1889.

Aeberli (80) publishes four cases of nuclear paralysis of the ocular muscles, three of which were probably due to changes in the blood-vessels and one to injury.

In Basevi's (81) case paralysis of the oblique muscles, of the superior and inferior rectus of the eyes, and finally of all the associated movements had been observed; the function of the other nerves of the head and neck was not impaired. The autopsy revealed chronic hydrocephalus with softening of the left thalamus opticus, and such a shrinkage of the corpora quadrigemina by pressure from above downward and from in front backward that they were scarcely recognizable. Comparing the disturbances of motility named above with the anatomical condition, and on the basis of seven experiments on rabbits, dogs, and cats, in which the disturbances of motility were noted after the removal of the corpora quadrigemina, the author concludes that although the centres of motion of the ocular muscles may be differently placed in the brain, there is nevertheless a common anatomical and physiological organ which must be considered the reflex centre and co-ordinator of those motions. This organ lies in the mid-brain, and is represented by the corpora quadrigemina.

Dantone.

Dufour (82) discusses in a lengthy paper the nuclear paralyses of the ocular muscles. After briefly speaking of the diagnosis of the seat of the pathological changes which cause paralysis of the ocular muscles, he takes up in particular the nuclear paralyses. He describes in detail the origin of the oculomotor nerve at the floor of the aquæductus Sylvii, and of the other ocular nerves.

Dufour's diagram of their origin is that of Kahler and Pick slightly modified. He reports on 220 cases of nuclear paralysis of the ocular muscles, partly in detail, partly very briefly. A discussion of them in a review of course is impossible. In regard to the etiology and treatment the paper contains nothing particular. In regard to the prognosis Dufour asserts that there is a form of nuclear paralysis which may readily disappear. Of 177 well-observed cases 110 took a chronic course or ended fatally, while 67 made a more or less complete recovery. In conclusion Dufour gives a very complete review of the bibliography and a table of the 220 cases. MARCKWORT.

GRANDCLÉMENT (83) describes as essential temporary paralysis of convergence a gradually increasing weakness of the latter during the adolescent period, which spontaneously disappears after about the eighteenth year. In opposition to his former opinion Parinaud believes that in the so-called insufficiency of the internal recti there is not insufficiency of the muscles themselves, but imperfect innervation for convergence. De Wecker calls attention to the insufficiency of the internal-recti muscles in myopia. Landolt declares that essential paralysis of convergence must be totally kept apart from muscular insufficiency. Meyer thinks that the insufficiency of the internal-recti muscles cannot be doubted. MARCKWORT.

JAVAL (84) publishes a case by which he endeavors to prove the influence of stereoscopic exercises upon the cure of strabismus. A tenotomy had previously been made upon both internal-recti muscles. MARCKWORT.

LAWFORD (85) reports on two cases of paralysis of the ocular muscles, which certainly was due to congenital syphilis. In both there was incomplete paralysis of the oculomotor nerve.

WERNER.

In a female patient with hereditary syphilis MEYER (87) found, besides bilateral ptosis, reflex immobility of the pupil, and absence of the knee-jerk, total ophthalmoplegia of both eyes.

STANDISH (88) reports five cases of partial tenotomies of the ocular muscles for insufficiency accompanied with asthenopia, mostly with satisfactory results. He thinks cases of insufficiency comparatively rare. In testing he thinks the power to overcome prisms deceptive, and relies more on the Graefe test. The use of prisms for a time after brings out the weakness more clearly.

BURNETT.

Wadsworth's (89) case was that of a boy aged fourteen, who held his head constantly to the right side and downward. He had never complained of asthenopia or bad vision, though in infancy his eyes had shown a tendency to roll about in a "wild" way. The left eye stood, during fixation when the head was in ordinary position, somewhat higher than the right; no stereoscopic vision in that position—images homonymous, the right higher; $V = \frac{15}{12}$; movements of eyes separately good. After tenotomy of the left superior rectus the faulty position of the head was relieved, though there is still some difficulty in obtaining stereoscopic vision. **Burnett.**

X.—ORBIT AND NEIGHBORING CAVITIES.

90. **Bock, E.** A cured case of caries of the orbital walls. *Allg. Wiener med. Ztg.*, 1890.

91. **Bull, C. S.** Contributions to the subject of tumors of the orbit and neighboring cavities. *Trans. Amer. Ophth. Soc.*, 1889.

92. **Cheadle.** Exophthalmic goitre. *Brit. Med. Jour.*, 1890, p. 19.

93. **Chibret.** Subluxation du globe de l'œil gauche dans le mouvement du regard en bas. *Progr. méd.*, 1890, No. 4, p. 71.

94. **Fumagalli.** Tumore orbitale. *Ann. di ottalm.*, vol. xviii., 6, p. 546.

95. **Galezowski.** Des affections syphilitiques du globe oculaire et de leur traitement. *Soc. d'opht. de Paris*, March 4, 1890.

96. **Lang, W.** Microphthalmus with cysts of the globe. *Royal London Ophth. Hosp. Rep.*, vol. xii., p. 289.

97. **Lawford, T. B.** A further note on cases of orbital sarcoma in children. *Royal London Ophth. Hosp. Rep.*, p. 329.

98. **Löw, L.** Contribution to the knowledge of enophthalmus. *Inaug. Diss.*, Berlin.

99. **Panas.** Considérations cliniques sur les abscès des sinus frontaux pouvant simuler des lésions indépendantes de la cavité orbitaire. *Arch. d'opht.*, vol. x., 3, p. 231.

100. **Schapringer.** Enophthalmus traumaticus ending in recovery. *New Yorker med. Wochenschr.*, June, 1890.

101. **Walker, C. H.** A case of distension of the frontal sinus with mucous polypi. *Royal London Ophth. Hosp. Rep.*, vol. xii., p. 351.

102. Williams, R. Remarks on orbital tumors from mucous distension of the frontal sinus, with cases. *Lancet*, 1890, p. 456.

Bock's (90) patient was a young shoemaker, æt. twenty-seven, in whom, probably in consequence of an injury received two years previously, caries of the walls of the orbit, with partial necrosis of these bones, had developed, extensive defects and a pathological communication between the orbital and cranial cavities being the result. The eye had been destroyed by panophthalmitis. After its enucleation, exenteration of the orbit was performed. After seven weeks the patient was discharged cured.

Bull (91) reports three cases of operation on tumors of the cavities close to the orbit. Case 1. Adeno-sarcoma of the lachrymal gland. 2. Abscess of the ethmoid cells, frontal sinus, and orbit, in a man of forty-six years, who had suffered long with naso-pharyngeal catarrh. The frontal sinus was opened and treated successfully. Case 3 was an unfortunate one of what was probably a myxo-sarcoma of the maxillary antrum, nasal fossæ, ethmoid cells, orbit, and cranial cavity, in a man of fifty-seven years, who had also been subject to naso-pharyngeal catarrh. The eye and portions of the tumor were enucleated for the purpose of contributing to the patient's comfort, which it did. He died comatose. Burnett.

Cheadle's (92) patient, a man, had shown marked symptoms of Graves' disease since 1868. The thyroid gland was so enlarged that it often caused dyspnœa. He continued to improve under tincture of iodine given internally, and remained perfectly well even after twenty years. The author thought from his observations that the tendency was to slow recovery in from one to five years. Werner.

Chibret (93) reports a case of subluxation of the eye. It occurred for the first time when the patient, a girl aged eight, stooped to pick up an object from the floor. She had previously had the whooping-cough. When the child stooped in Chibret's presence subluxation at once took place, but the eye went back spontaneously as well as upon slight pressure. After wearing a compressive bandage for twenty-five days the child recovered, no further subluxation taking place. Marckwort.

Fumagalli (94) successfully removed a tumor 4 *cm* long and 2 *cm* thick from the lower inner corner of the orbit, with preservation of the globe and sight. Dantone.

Galezowski (95) describes a case of exostosis in the region of the right eyebrow and forehead in a boy aged nine years. As he came to the conclusion that hereditary syphilis was the cause, he gave iodide of potassium in 2- to 3-grm. doses daily and began inunction, from which, however, he was obliged to desist after two weeks on account of salivation. After the prolonged use of iodide of potassium the exostosis disappeared.

Marckwort.

Lang (96) describes the anatomical appearances of a congenital cyst of the lower lid associated with microphthalmus. The other eye was normal. On excision it was found that the cyst was adherent to the small eyeball at the lower border of the optic nerve, its thin wall was continuous with the sheath of the nerve behind and with the sclerotic in front, and its cavity communicated through a narrow neck with the interior of the globe. The choroid terminated at the neck of the cyst, while the retina extended a short distance over it. The lens was large in proportion to the eyes, and brown in color. There was also a coloboma of the iris below.

Werner.

After a discussion of enophthalmus and its causes, Löw (98) reports two cases which were due to an injury. In both of them the optic nerve became pale, indicating an injury to the orbit.

Panas (99) saw in the course of a year four cases of abscess of the frontal sinus, and calls attention to their diagnostic aspect, as none of them had been recognized by others. In all cases of suppuration from the upper edge of the orbit there may be an affection of the frontal sinus, the extent of which may be very great, so that abscesses and fistulas may appear in the middle and even at the outer edge of the entrance to the orbit. The absence of ozæna does not speak against an affection of the sinus. *Frontal headache* is characteristic ; it is never absent, and is explained by the abundance of fibres of the fifth nerve in the mucous membrane of the frontal sinus. In treating the disease the author makes an incision directly below the eyebrow and another perpendicular to it upon the base of the nose. The triangular flap thus formed is detached from the bone together with the periosteum, and the bone is then trephined. The further treatment consists in the irrigation with a solution of bromide of mercury 1:20,000 and introduction of a drainage tube. From his studies on corpses he thinks it is possible to extend this drain into the nose. He has not yet tried which method produces more rapid results, but a cure always requires months. No cerebral symptoms have thus far been observed. v. MITTELSTÄDT.

SCHAPRINGER (100) reports that after a fall upon the right temple ptosis of the right upper lid, enophthalmus, and diminution of the tension of that eye developed in a girl aged seven years. After four days this condition had completely disappeared. He thinks it may have been due to paralysis of a peripheral branch of the sympathetic nerve of the head, caused by pressure of a small hemorrhage.

WALKER'S (101) patient, a boy aged eighteen, had received an injury when three years old which left a scar on the brow. When first seen he had proptosis due to abscess in the roof of the orbit, which pointed at the outer end of the eyebrow. It disappeared, but recurred twice; the third time, a hole was discovered in the orbit through which a large polypus protruded, which was removed. Course of healing smooth. WERNER.

WILLIAMS (102) alludes to the difficulty of diagnosis of tumors of the orbit before fluctuation has appeared. WERNER.

MISCELLANEOUS NOTES.

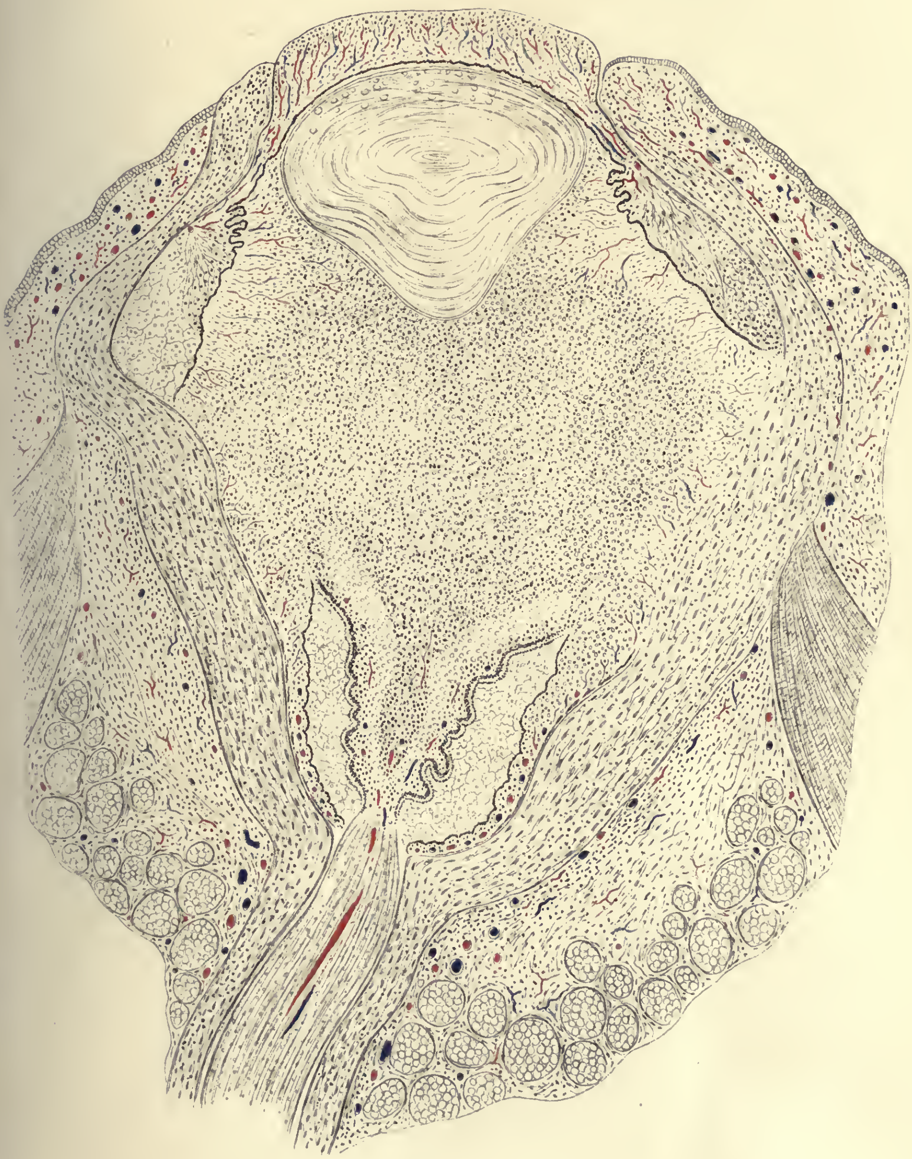
C. A. Coccius, born in 1825, died November 24, 1890. He studied in Leipzig, Prague, and Paris. From 1849 to 1857 he was assistant to the chair of ophthalmology (Ruete) and lecturer at the University of Leipzig; from 1857 to 1867 he was extraordinary, and from 1867 until his death ordinary, professor of ophthalmology and executive surgeon of the University Eye Hospital in Leipzig. His youth fell in the initial period of ophthalmoscopy, when every look through the wonderful instrument was a discovery. Helmholtz's first publication, "Description of an Eye-Mirror," in 1851, was followed by Ruete, who introduced the concave perforated mirror in 1852, in a large and rather unwieldy form, which Coccius replaced by a handy instrument, a plane mirror with a convex lens as reflector. This instrument gained a certain popularity, and v. Graefe used it all his life.

Coccius enriched the theory and practice of eye-surgery by many very important discoveries, of which we mention: the venous pulse on the optic disk; the ophthalmoscopic appearances of the fundus oculi, especially of the yellow spot, with the reflex of the fovea; the determination of the seat of opacities in the media of the eye. Among his numerous scientific contributions the following are most valuable: On the Nutrition of the Cornea (1852); On Glaucoma, Inflammation, and Autophthalmoscopy; A Simplified Ophthalmometer (the doubling of the images produced by a tourmaline plate); several papers On the Mechanism of Accommodation.

Coccius was an original thinker and a fertile investigator whose name occupies a prominent position in the first period of ophthalmoscopic discoveries. His therapeutic, in particular his operative, peculiarities were not always felicitous. He was a successful practitioner, a popular teacher, and a kind man.

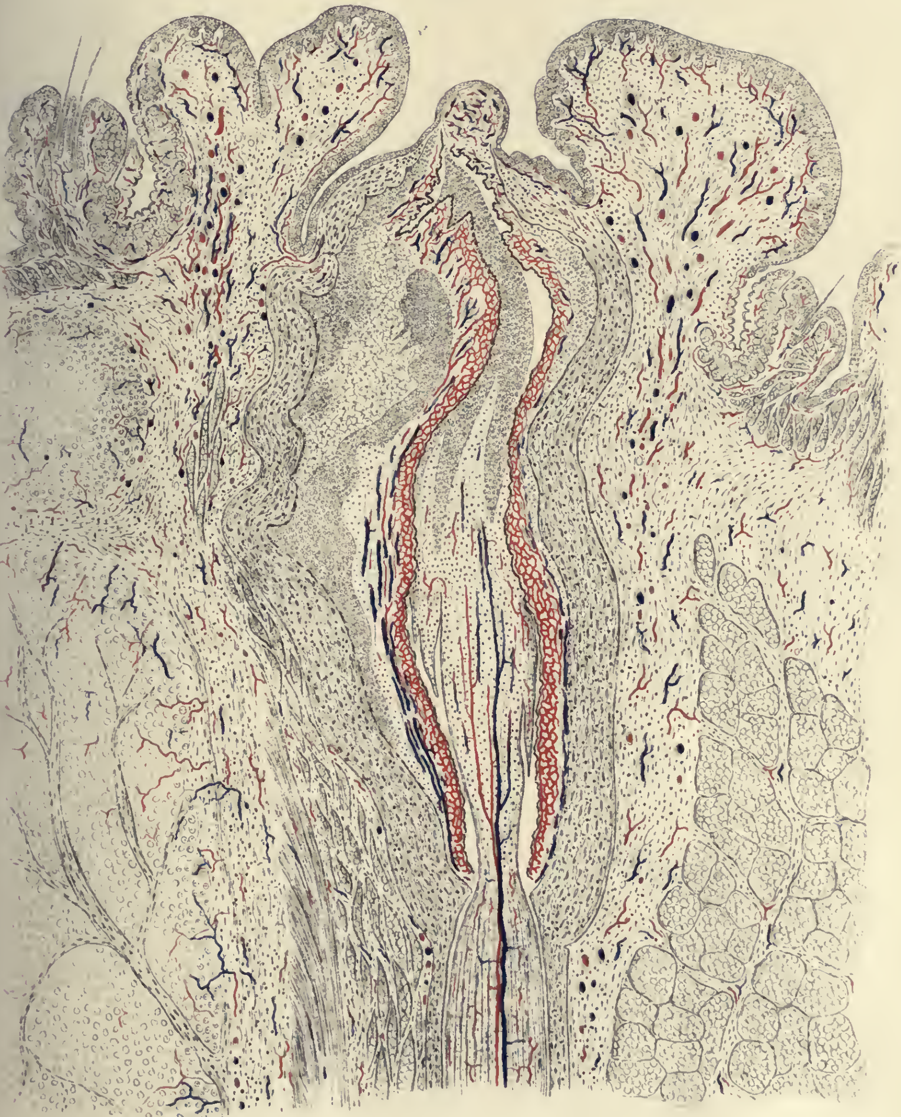
H. K.

Fig. 2.



Prof. Schöbl, del. C. Krapf, München, lith.

Fig. 3.



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VOL. XX. No. 2

ARCHIVES OF OPHTHALMOLOGY.

ON THE DEVELOPMENT AND COURSE OF THE MEDULLATED FIBRES IN THE CHIASM OF THE OPTIC NERVES IN MAN.

By Dr. STEFAN BERNHEIMER,

ASSISTANT TO THE UNIVERSITY EYE-CLINIC IN HEIDELBERG.

Translated by Dr. Ward A. Holden, New York.

(*With plate x. of vol. xx., Germ. ed.*)

IN examining a series of human embryonic eyes some years ago, I was struck by the fact that in the optic nerve, some millimetres distant from the lamina cribrosa, the fibres appear as simple axis-cylinders without a medullary sheath. Even in the eyes of infants born at term, which have died at birth or shortly after, no medullary substance is found in this location, but naked axis-cylinders alone, such as are found in adults only anterior to the lamina cribrosa. This fact, not mentioned in the literature, led me to study the matter thoroughly, and to follow more accurately the development of the medullary sheath in the fibres of the optic nerve and chiasm in man, as far as my limited material would permit, with a view of determining when and where the axis cylinders of the conducting apparatus of the retina first take on their medullary substance.

The time and place of the first development of medullary substance were determined with certainty, and the successive stages of development were followed in their order from the optic tract through the chiasm to the lamina cribrosa.

The question whether there exists a total or a semi-decussation of the fibres of the optic nerve, after having been discussed for decades, remains to-day unsettled. The most varied methods of investigation have been employed to prove the one or the other theory, and we should certainly welcome a new method which would enable us to explain the many direct contradictions of authors, and the various sources of error in other methods, and finally give us the purely anatomical proof, which still fails, of the existence of the uncrossed bundle.

All these requirements would seem to be met by our method, which consists in following the formation of medullary substance in the retinal conducting apparatus in man, from its earliest beginning to its full development, by means of Weigert's method of staining.

The solution of the following questions will be attempted in this paper:

1. When and where in the retinal conducting apparatus do the naked axis-cylinders take on their medullary sheath?

2. What is the course of the medullated fibres in the chiasm of the embryo and infant?

I.—DEVELOPMENT OF THE MEDULLATED FIBRES.

In determining the time and place of the first formation of medullary substance a great number of human embryos were studied, which, after the cranium had been opened, were placed as early as possible in Müller's fluid, in which they remained from six weeks to several months. Most of the embryos of the second third of intra-uterine life had lain in Müller's fluid longer, as they had been collected earlier for another purpose by my colleague, Dr. C. Hess, who kindly put them at my disposal. For the remainder of the material, consisting of aborted embryos, new-born and young infants, I am indebted to the Heidelberg Pathological Institute, Professor Weigert in Frankfort, and Dr. Winter in Berlin. The age of most of the embryos was known, and the age of the others was computed from their length. The youngest measured 12–13 *cm*, and from this the series extended to infants in the second, third, and fourth weeks of extra-uterine life.

After lying in frequently changed Müller's fluid for not less than four weeks, the specimens were placed in alcohol sixty per cent., and later eighty per cent., until the alcohol, which was changed daily, showed no longer a yellow color, the vessels remaining during this entire period in the dark. After being left for twenty-four hours in absolute alcohol, the specimens were imbedded in celloidin in the usual manner. They should not be brought from Müller's fluid into water first, for the water not only extracts the excess of chromic acid, but probably also redissolves a portion of the reagent which has penetrated into the tissue, so that it can never be said with certainty whether a negative result from Weigert's stain is to be ascribed to the actual absence of a medullary sheath or to the incomplete hardening of it. When the specimens are not put into water, but are immediately rehardened in alcohol in the dark, the slightest traces of medullary substance will be shown by Weigert's stain.

Dispensing with water and treating the specimens with alcohol has the additional advantage that the precipitates, which otherwise often confuse the stained preparations, are not formed. These niceties in technique are well known, it is true, but they need to be expressly emphasized, as their observance is of very great importance in such investigations.

The specimens, after being imbedded, are placed in neutral acetate of copper solution in an incubator for forty-eight hours, then cut with a microtome in thin serial sections, stained with Weigert's hæmatoxylin for twenty-four hours, washed quickly in distilled water, and carefully decolorized. It is well to dilute the differentiating fluid, as this prevents a too hasty decolorization, and the more gradual process can be better controlled. The sections removed from the differentiating fluid are put into water, in which they may remain for hours, and then for a few minutes into alcohol, 90%. They should not be left long in the latter, as they become pale, and traces of medullary substance or delicate young medullated fibres may easily be overlooked. Finally, after being cleared in origanum oil, the sections are mounted in Canada balsam.

A marked variation in color, in sections from specimens which are fresh or which have been hardened in Müller's fluid, is observed only after the formation of medullary substance is far advanced. During the whole of embryonic life, and even in infants dying at the time of birth or a few hours after, there still predominates that indefinite, hyaline, gray to reddish-brown color of the soft, almost jelly-like tissue.

Before the twenty-fifth week of embryonic life no clearly recognizable chiasm appears. In the early periods no medullary substance is found. The axis-cylinders are developed but are small. Actual connective-tissue fascicles are not clearly recognizable between the bundles of axis-cylinders, but faintly stained connective-tissue corpuscles with processes and well-marked nuclei are seen arranged in rows, and between these there are indications of connective-tissue fibres. In this young tissue great numbers of the finest capillaries are found, which run longitudinally in part, and in part form transverse anastomoses. In reality a capillary network exists, through which the finest axis-cyinders pass.

Connective-tissue septa form a close network through the optic nerve in its entire course. These septa are very rich in apparently free-lying cells, to which as well as to the numerous capillaries they owe their vitality. At some points these spindle cells seem to be in connection with the walls of the finest capillaries, and are therefore to be considered vascular sprouts.

Lack of material between the eighteenth and twentieth and the twenty-eighth and thirtieth weeks of embryonic life made it impossible, unfortunately, to follow the various form-elements in the chiasm through every stage of their development.

In all the specimens examined, the chiasm together with the tract as far as its free root and the nerve as far as the optic foramen were cut in horizontal serial sections.

Sections of the chiasm of an embryo of the twenty-eighth to the thirtieth week, stained by Weigert's method, appear of a uniform pale-yellow color when slightly magnified, and show a delicate longitudinal striation, which in the middle of the chiasm becomes more netlike. Running in the same general direction with these delicate striæ are seen dark-brown or black stripes, which, while interrupted in many places, are on the whole regular. The substance connecting the two inner longitudinal sides of the tract and the posterior angle of the chiasm is of a fairly uniform bright yellow color, shows no striation, and is somewhat granular.

Under a higher power the finely striated ground-substance of the tract resolves itself into sharply defined single fibres, which run beside each other, interlacing to some extent, but always pursuing a direction parallel to the long axis of the tract. The individual fibres do not unite to form bundles. They are separated from each other by a clear homogeneous cement-substance, and present the appearance of unmedullated axis-cylinders.

In the chiasm the individual axis-cylinders are similar to those in the tract, but the mass presents a different appearance. The fibres cross each other irregularly, and this entanglement is greatest in the central portion of the chiasm, and much less marked near the angles.

In the nerve, the picture is again changed by the characteristic connective-tissue septa, which here, as in the developed nerve, spring from the pial sheath and divide the mass of axis-cylinders into small bundles which partially interlace.

The black stripes seen in the tract, chiasm, and nerve under a low power now appear as fine capillaries, which run through the whole preparation in every direction, but more particularly in the direction of the bundles of axis-cylinders. The great number of leucocytes, in comparison with the small number of red corpuscles, is striking.

Besides these cells, the round or oval sharply contoured black or dark-yellow cells, described above, are found free in the interfibrillar substance, and I am inclined to regard these as belonging to the connective-tissue cells.

Capillaries are found everywhere in the yellow finely granular ground-substance between the tracts, just as they are found in the tracts and chiasm, and there are also seen large dark-yellow or black round or oval cells, with a large round nucleus and a large clear protoplasmic body. These are the ganglion cells described by J. Wagner[6] and Meynert,[7] which Meynert considers ganglia of origin of the nerve fibres which pass to the nerve of the same side; he therefore calls this region the basal opticus ganglion.

At the thirtieth week of embryonic life, the chiasm under a lower power presents a picture similar to that found in the other case. It is difficult, in the entanglement of pale fibres, to recognize with certainty the continuation of any particular fibre. At this time a substance begins to appear about the naked axis-cylinders, of a different chemical composition from them, and distinguished by its dull-gray color. In other parts a number of swellings appear on a single axis-cylinder, while the intervening portions retain their original color. In almost all the sections of the chiasm in this embryo of the thirtieth week, such points were found scattered about, but never affecting more than a small portion of the axis-cylinder, and then only in the tracts and chiasm, never in the nerves.

Two embryos of about equal development were examined, and the two presented a similar picture. The beginning of the formation of medullary substance was seen in both to the same limited extent, without a trace of medullary substance being found in the nerve. This speaks for the constancy of the condition observed.

Sections of a chiasm of the thirty-second to the thirty-third week presented a very different picture. Even under a low power, in certain places in the preparations, besides the darkly stained capillaries and free blood corpuscles, fine darkly-stained fibrils could be seen between the delicate yellow striæ of the axis-cylinders. Under a higher power two things are seen: first, narrow bundles of axis-cylinder fibres, which have the same dull-gray color that was seen in the axis-cylinders in the sections described above, and are as short as those, having two or three delicate swellings, and passing over, for the most part, into naked axis-cylinders; second, near the first, dark-blue sharply contoured fibres, clearly thicker than a naked axis-cylinder, and having also a number of swellings of varying size throughout their whole extent. The greatest number of the medullated fibres is found in the tract, but scattered and not confined to any particular region. They become rarer nearer the nerve, and are not found at all in the neighborhood of the optic foramen.

In the region of the gray matter which bounds the posterior angle of the chiasm, black arched fibres are found, which are no doubt the medullated fibres of Meynert's commissure, that runs in an arc in the gray matter along the two tracts and the posterior angle of the chiasm. The preparations now to be described were from an eight-months' fœtus which died seven days after birth. It was thus of about the same age as the embryo of the thirty-second to the thirty-third week just spoken of, with the difference that the last week had not been passed in the uterus.

Macroscopically nothing characteristic was found, but microscopically a marked difference between this and the previous specimens was noticed. Under a low power all the sections of the chiasm showed a far more extensive black striation than was found in sections from the previous case. Under a higher power a corresponding increase was seen in the number of medullated fibres in the tracts, chiasm, and nerves. While the optic nerves of the unborn fœtus of the same age showed only traces of medullary formation here and there in their lower portion, in this case the medullary substance was in an advanced stage of development. In the lower portion of the nerves, the axis-cylinders were at least as fully covered by a medullary sheath as those of the tract in the previous case. The anterior angle of the chiasm in some preparations has delicate medullated fibres running in a commissural direction. Meynert's commissure is especially rich in medullated fibres, which here show the most advanced development not only in number, but also in the appearance of the individual fibres, which are thicker than the medullated fibres of the tract and chiasm in the same individual. The angles of the chiasm have more medullated fibres than the interior. Even in the new-born infant the medullary substance of the nerve is much more delicate than that of the tract, and does not extend forward to the lamina cribrosa.

The chiasm of the new-born infant is to be considered a nervous organ incompletely provided with medullated fibres, for while the greater number of axis-cylinders in the tract, chiasm, and nerve have a medullary sheath, there are still a certain number of naked axis-cylinders scattered among the medullated fibres. The organ is also incompletely developed, since the formation of medullary substance does not extend to the lamina cribrosa, its peripheric limit in the adult.

The youngest individuals studied that had enjoyed extra-uterine life were infants from three to four weeks old.

In these, the medullary sheaths in the nerves extended to the lamina cribrosa. Sections through the chiasms of these subjects presented a remarkably fine and clear picture, which will be described in greater detail later. All the axis-cylinders, as far as could be told with certainty, exhibited medullary sheaths, which appeared young, however, and, compared with those in the adult, were thinner and more delicate, and showed a greater number of swellings. Besides this, free cells are still seen in the interstices of the chiasm, which are formed of apparently structureless interstitial substance (neuroglia). The delicacy of the medullary sheaths makes the topography of the section clearer. As the sheaths have not the thickness which they have in the adult chiasm, an appreciable interspace exists between the individual fibres, and since the general network of the medullated fibres is a loose one, the individual fibres are, as it were, isolated. In sections of the adult chiasm, on the contrary, the fibres have a thicker, less delicate sheath, with very small swellings. On account of this increased thickness of the sheaths in the adult chiasm, the individual fibres are so pressed together that the free spaces between them, so clearly seen in infancy, either vanish entirely or become reduced to a minimum, so that it becomes impossible to follow single fibres distinctly or to isolate them in the chiasm. This may be said to be the chief characteristic of the young chiasm.

The facts discovered with reference to the manner and the time of the first development of the medullary sheaths in the chiasm, may be briefly stated as follows:

No trace of medullary substance can be found in the chiasm or in its roots and processes before the twenty-ninth week of embryonic life. At this time the first beginnings of its formation are seen in the delicate swellings on the axis-cylinders, and these stain dark and often show dark-stained processes on both sides of the axis-cylinder. The processes from two such swellings on a fibre may often be seen to join.

In the thirty-second to the thirty-third week, swellings and their prolongations are seen on a far greater number of fibres, and these portions are stained dark. These swellings are more numerous in the fibres of the tract, and become rarer toward the nerve, ceasing altogether in the nerve trunk. The most marked changes in the chiasm are in the two lateral and the posterior angles, the position of Gudden's commissure and the so-called anterior commissure.[1]

In an embryo of equal age, where the last week of life had been extra-uterine, the influence of this extra-uterine life on the development of medullary substance after the thirty-second week, previously pointed out by Flechsig, was confirmed by the microscopic proof of the more advanced development of the medullary sheaths. This case exhibited the transition to the mature fœtus, in which the tract and chiasm have a greater though not the full amount of medullary substance. The lack of complete development is shown not only by the thinness and delicacy of the medullary sheaths, which are just visible under a low power, but also by the presence of a number of still naked axis-cylinders, and by the fact that the medullary substance ceases some distance behind the lamina cribrosa, while the ciliary nerves at this point in the same eye are made up almost altogether of medullated fibres.

The fibres of Meynert's commissure in the gray matter of the tuber cinereum show the most advanced development in the embryo of the thirty-second to the thirty-third week, and this advanced development continues throughout the succeeding stages of embryonic growth.

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[1] Michel,[8] in a child in the thirty-second week of uterine life, dead seventeen hours after birth, found the medullary sheaths well developed in the fibres which form Gudden's commissure at the dorsal margin of the chiasm and the tracts. Very delicate medullated nerve fibres, recognizable by their pale gray color, were also seen in the nerves and chiasm. The number of these fibres was greater in the tract, and the extreme lateral bundles in the chiasm seemed to have more medullary substance.

In the chiasm of the infant of the second to the third week, all the axis-cylinders possessed a medullary sheath in their whole extent; the young chiasm is distinguished, however, by the fact that the medullated fibres are thinner and more delicate than in the adult, and that the meshwork, as far as it may be considered such, is much looser, permitting the naturally isolated fibres to be more readily followed in their course.

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II.—COURSE OF THE MEDULLATED FIBRES.

The course of the medullated fibres in the chiasm in man has long interested physiologists, anatomists, and ophthalmologists.

In 1826 Johannes Müller,[1] without anatomical grounds, at a time when there were no means of following the course of single nerve fibres, advanced an hypothesis which attempted to explain the problem of single vision by assuming the existence of identical points in the two retinas. He suggested that each root-fibre of the tract might divide in the chiasm, and the two branches end in corresponding points in the two retinas. Two years later he saw that this hypothesis was untenable, for it had been discovered that the fibres pass undivided through the chiasm, and that, therefore, the combination of the impressions must take place through some special arrangement in the central organ proper. Each root of the chiasm must then carry the fibres from the corresponding halves of both eyes to the brain. He assumed that in man and animals, where the horopters coincide in part, the outer fibres of the tract pass to the nerve of the same side, the inner fibres to the nerve of the opposite side, and he thought also that he had seen commissural fibres between the peripheric and central origins of the nerves.

Two years before this, Treviranus,[2] studying the monkey's chiasm hardened in alcohol and exposed for a time to the action of caustic potash, found that after the sheath had been removed and the fibres separated by means of fine needles and a brush, the fibres in the upper outer portion of the nerve appeared to pass from the brain to the eye without anastomosing in the chiasm with the fibres of the other nerve; while the fibres of the inner lower portion of each nerve passed over in the chiasm to the nerve of the opposite side, and became intermingled with its fibres.

F. Arnold[3] arrived at similar results after isolating the fibres in the human chiasm, and the drawings in his famous "Tabulæ Anatomicæ," 1839, were apparently conclusive.

In 1831 Hilderbrandt,[4] in his "Anatomy of Man," presented a number of observations of a pathological nature, supporting the idea of a partial crossing, and he was followed by Longet,[5] 1842; Foville,[6] 1844, and Cruveihlier,[7] 1845. The first spoke of the macroscopic appearance in a case of unilateral atrophy of the optic nerve, where the difference in thickness in the two tracts did not indicate a total crossing. The others studied also the course of the fibres, and Cruveihlier, while he did not mention an anterior commissure, described a posterior one, which was probably that discovered later by Meynert.

The first investigations of importance concerning the course of the fibres in the chiasm were made by Hannover[8] in 1852. He found that when the chiasm and tract were left some months in a weak chromic-acid solution, the separate bundles of fibres could be isolated.

Experimenting with this method, Hannover distinguished the following bundles: (1) A commissura ansata, composed of fibres which descend from the lamina terminalis cinerea, situated before and above the chiasm, and which run superficially first on the anterior and later on the inferior surface of the chiasm, and are finally lost posteriorly in the tuber cinereum and the infundibulum. (2 and 3) A fasciculus sinister et dexter, which run, one on each side, mostly in the outer border of the chiasm; they are also seen on the upper and lower surface of the chiasm, but do not reach to the middle line. Passing forward, the fibres are directed outward and downward, the upper fibres in the tract becoming thus gradually outer fibres, and further forward toward the chiasm the outer fibres become lower fibres. (4) A commissura arcuata anterior, forming an isolated arched bundle $\frac{3}{4}'''$ thick, which lies on the anterior concave margin of the chiasm, and sends a branch forward in the inner border of each optic nerve. The greater number of the bundles lie near the upper surface of the chiasm. Erdl[9] had already called attention to this bundle in 1843. (5) A commissura arcuata posterior, which lies superficially at the posterior concave margin of the chiasm, and forms an isolated bundle. As the fibres run from the brain to the chiasm, they pass upward, and thus take part in the turning of the fasciculus dexter and sinister. (6 and 7) A commissura cruciata, which, next to the commissura ansata, contains the smallest number of fibres, and is but $\frac{1}{2}'''$ thick. One bundle passes from the right side to the left optic nerve, and the other passes in the opposite direction. It lies in the interior of the chiasm, apparently nearer the upper surface. This commissure is very difficult of demonstration, because of the intimate blendings of the bundles and fibres. All these grosser divisions are made up of smaller bundles, which lie near each other without mingling, except in the case of the commissura cruciata. These discoveries of Hannover's were verified in 1854 by Sohmen[10] by means of microscopic sections, and the question seemed to be settled.

But, six years later, a paper was published by Biesiadecki[11] on "The Chiasm of the Optic Nerves in Man and Animals," in which methods of investigation, similar to those of Hannover, lead to results almost contrary to his. The work had been performed under Brücke's direction, and this fact gave it special weight. It closed with the words: "After my investigations, I must agree with those who have supposed that a complete crossing of the optic fibres takes place in the chiasm in man and in all vertebrates."

His method was, first, the microscopic examination of sections from specimens hardened in chromic acid; secondly, the examination of teased chiasms hardened in chromic acid and laid in concentrated potash solution, whereby the connective tissue between the nerve bundles became partly more transparent, and partly dissolved, so that the bundles could be separated without difficulty. Finally, chiasms of men and animals blind in one eye were examined in a similar manner. In man he found that the commissura ansata of Hannover existed, but was composed, as Valentin[12] had said, of tendinous fibres. "On the upper surface of the chiasm the fibres are seen to run continuously from the nerves to the posterior angle of the chiasm; the lower fibres of the nerve pass on the upper surface to the opposite root, the outer fibres, however, cross first in the angle and run in the root from inward, outward, crossing the other fibres of the root. In some chiasms I found a striking departure from this rule, in that the fibres from the nerve extended in a process becoming ever thinner, having an inner convexity and an outer concavity, and ended in a point at the side of the posterior angle at the chiasm. The fibres of these processes seemed to remain on the same side." The lower surface is similar to the upper in that the same process takes place, but in a reverse order. If the nerve bundles are removed separately from the chiasm, well preserved specimens give pictures which show the complete crossing of the root fibres to the opposite nerves. Preparations less well preserved often show bundles which seem to remain on the same side, but when these are examined with the microscope, they are seen to be crossed fibres, which have been torn off after the crossing. The fibres which seem to run in a commissural direction also reveal themselves to be fibres crossing in a slightly curved arc.

Brown-Séquard's[13] studies were in the direction of experimental pathology, and depended upon the cutting through of the chiasm of animals in the median line, after which total blindness resulted. This indicated a total crossing, but could not strictly be considered as holding good for man.

In 1873 Mandelstamm[14] repeated the experiments of Biesiadecki on the human chiasm, but isolated the bundles more in layers, so that the course of the fibres could be followed in different planes.

" In following the fibres of the tract in the upper surface of the chiasm, the extreme outer ones are seen to pass to the outer angle, and there turn to the lower surface and cross. This relation is best seen in chiasms with acute outer angles. These are the fibres which in sections are seen to cease at the outer angle of the chiasm, as if cut off, and seem not to exclude the possibility of the existence of a lateral commissure."

After removing the upper layers, fibres are found in the middle layers which describe an arc. " These fibres of the middle layers of the chiasm, because of their arched course, may simulate anterior and lateral commissures, especially the latter. The isolation of the bundles will alone show the actual relation. Their horizontal sections are also of value, and those especially from the upper surface exhibit a total crossing of the fibres."

The same year Michel[15] employed horizontal sections, and his conclusions were that in the chiasm of man all the optic-nerve fibres cross, and no trace of commissures can be found within the chiasm.

Michel's results found a speedy confirmation in a paper by Scheel,[16] who used not only horizontal sections, but also divided the chiasm in a sagittal plane, and then made wedge-shaped sections from the left half, the base of which came from the anterior angle of the chiasm, and further from the left nerve. The right half was cut in a similar way. In spite of this complicated proceeding, he came to the conclusion that all the fibres cross, and that no commissures exist. He verified by this method the existence of fibres which spring from the tuber cinereum and the lamina terminalis cinerea and run in the upper and lower surface of the chiasm, but not in the chiasm itself, and he acknowledges that these fibres pass in part to the side next them and in part form the commissura ansata.

The contributions which Gudden[17] then made to this very involved subject were of great importance. He brought the experimental method of investigation in animals to the highest degree of perfection. He also studied the human chiasm in horizontal sections, yet, as he says, this must be rightly done. Definite results can be obtained only by following section for section, bundle for bundle. "Michel does not seem to have done this. His drawings are correct, but not convincing. If he had had a complete series of sections, he might have made just as beautiful drawings to prove that the crossing is not complete, and that the view of Hannover-Henle,[18] that a lateral direct fascicle exists, is correct." Gudden denied the existence of an anterior commissure. He supported the idea of a direct bundle, but added that the fibres which cross lie mostly in the lower half of the chiasm, and those which do not cross, in the upper half.

Gudden also measured transverse sections of the tracts and nerves in dogs where one eye, or both, had been enucleated, and did this also in human chiasms where unilateral atrophy had existed. These measurements confirmed the idea of a partial crossing. All of Gudden's results were verified by M. Reich, in the *Centralbl. f. med. Wissensch.*, 1875, p. 480.

Adamük[19] produced unilateral atrophy in new-born cats and dogs by enucleating one eye. Study of the chiasms and tracts some months later showed that the crossing in these animals is not complete. In addition, he gave a macroscopic description of two human chiasms where one eye had been lost in infancy. The measurement of transverse sections of the nerves and tracts gave results which could only be explained by the assumption of a semi-decussation.

Purtscher[20] studied the nerves, chiasms, and tracts in six cases of unilateral atrophy of the optic nerve, and three cases of bilateral atrophy. The eyes in most of the cases had been blind for years. The sections of the six cases of unilateral atrophy verified all of Gudden's important results. Two cases of bilateral atrophy showed the commissural system discovered by Gudden, *i. e.*, the posterior commissures of Gudden and Meynert, as an isolated bundle of normal fibres in the otherwise totally atrophic tracts. The sections were studied partly in glycerin or solution of caustic potash, partly stained with carmine after previous treatment with palladium chloride.

Baumgarten's[21] microscopic examinations of human chiasms with unilateral atrophy were not less conclusive of a semi-decussation.

Stilling[22] in 1882 resumed the former method of isolation of the bundles, but macerated and dissolved the connective tissue by means of alcohol and pyroligneous acid. His results favored the idea of a partial crossing.

A series of investigations of pathological cases now follow (Marchand,[23] Richter,[24] Nieden,[25] and others), in which unilateral or bilateral atrophy existed in consequence of the loss of one or both eyes. These were examined microscopically in part, macroscopically in part, or the measurements of the transverse sections of the nerves and tracts were compared (Deutschmann,[26] Samelsohn[27]). The results indicated a semi-decussation.

Kellermann's[28] investigations are worthy of special notice, as he treated his sections of the chiasms with two reagents hitherto unused—gold and osmium. From his examination of the chiasm in a case of unilateral atrophy, he was led to regard the crossing as incomplete. He did not accept the view, however, that the direct bundle runs separately by itself, but that fibres scattered everywhere through the nerve pass into both the crossed and uncrossed bundles. The case was not altogether convincing, as the nerve on the side where vision remained showed a partial atrophy.

The numerous experiments on animals, first made by Gudden[29] and imitated by his pupils and others, are interesting in a high degree, as they are conclusive of the fact that there is a semi-decussation in a number of mammals, such as the rabbit, cat, and dog.

We shall not go into a detailed discussion of these experiments, as this had been done a number of times by others, and for the further reason that here where the question is in regard to the course of the fibres in man, these experiments have no direct application. It is not going too far to suppose, however, since the higher developed mammals show a more complicated arrangement of nerve fibres indicating a partial crossing, that this is probably the case in the mammal of highest development—man. This is, at any rate, an analogy more justifiable than that of Michel, who says: "Since a complete crossing occurs in mammals, it would seem strange if this were not also the case in man."

This is, however, not the case either in man or in the higher mammalia. Gudden,[30] who in 1875 was of the contrary opinion, proved this experimentally for the rabbit in 1879. In his first experiment, after the removal of the posterior portion of the cerebral hemisphere in a new-born animal, the whole base of the brain with the tract was taken out. Six months later examination showed that the injured tract was completely atrophied, the nerve of the same side and the opposite tract were normal, and in the apparently atrophic opposite optic nerve, a thread-like normal direct bundle could be seen after being stained with osmic acid. In the second experiment the frontal bone was removed and one half of the chiasm taken out with a sharp spoon. Examination of the fully developed animal showed nothing in the chiasm excepting the direct bundle of the one side, which could be followed from the optic nerve into the tract of the same side.

These proofs have not satisfied Michel, either of the semi-decussation in mammals or of the semi-decussation in man, which might naturally be supposed to follow from the other.

In an elaborate brochure with fine plates, published in honor of Kölliker's seventieth birthday, Michel[32] repeated his well-known views on the crossing of the fibres in the chiasm of birds and mammals, including man, and hoped thereby, he says, to convince others, not of the theory but of the fact of the total decussation in the chiasm. The main point, which was new in his manner of investigation, was the use of Weigert's method of staining for the medullary sheaths.

He studied new-born and full-grown owls, guinea-pigs, rabbits, cats, and dogs, and some preparations from man. Serial sections were made with a microtome in a horizontal plane, so that nearly every section included the chiasm, both tracts, and both nerves ; transverse and longitudinal sections of the nerves and tracts were also made. The owl's chiasm was obtained from a bird in which one eye showed an opacity of the lens and an atrophy of the nerve of the same side. In the other animals one or both eyes had been enucleated for the purpose of determining the relations of the commissure. In the rabbit some chiasms were divided in a median plane. The examination revealed in every case a total crossing of all the fibres of the tracts.

Besides these, three cases of unilateral atrophy of the optic nerve and a case of unilateral anophthalmus in man were examined. These cases also showed a complete decussation of all the fibres of the tract.

Let us look more closely at the report of these four cases in which the human chiasm was examined.

The first case was that of a man of sixty-seven, who had lost his right eye in early youth. In the whole series of horizontal sections the right optic nerve is seen to contain, in a yellow finely fibrillar tissue, a great number of dark-colored nerve fibres of varicose appearance, which run not in isolated bundles, but as single fibres. A lighter color is seen only in the dorsal portion of the left half of the chiasm, and especially in the dorsal portion of the left tract. The light point in the dorsal portion of the left half of the chiasm shows the interweaving of the fibres as in a straw mat, and the extreme lateral bundle of the right nerve bends in a fairly convex curve toward the tract of the same side before it passes to the opposite side. In the dorsal portion of the left tract a narrow atrophic bundle is seen, directly continuous with the atrophic bundle in the chiasm.

The second case was that of a man of sixty-six, who had lost his left eye forty-six years before. The whole series of horizontal sections showed a marked thinning of the left nerve and the right tract. The tract of the same side shows a normal thickness, and is composed solely of normal bundles of nerve fibres. The degeneration in the atrophic left nerve is general.

The third case was that of a girl of fifteen, whose right eye was removed on account of gliosarcoma. Five years afterward the left eye showed a choked disc, and death occurred a year later. Nearly the entire left hemisphere of the cerebellum is occupied by a cyst filled with serous fluid ; the lateral ventricles, especially the left, are markedly dilated and filled with fluid ; the right optic nerve is very thin, the left tract is less prominent, but completely white.

" The whole series of horizontal sections shows the right nerve totally degenerated and diffusely yellow, with here and there in some sections a short, darkly stained fibre. The right tract in its entire breadth shows narrow varicose bits of stained nerve fibres, with occasional fibres of larger calibre mingled with a number of bits of medullary substance. The changes are most marked in the dorsal portion, and in some sections in the middle portion, so that a finely fibred yellow tissue presents itself, in which bits of nerve fibre are seen. The left nerve shows degenerated nerve fibres scattered uniformly over its entire breadth, the degree of degeneration however being less than in the opposite tract. The degeneration is again much more marked in the left tract than in the right. In the central portion of the chiasm there is a regular interweaving of completely degenerated fibres and such as show a moderate degree of atrophy."

The fourth case was that of a child with unilateral anophthalmus, who had died when five months old. A carmine stain was unfortunately used. " I can only say that the transverse sections of both tracts showed a normal relation, and that the right nerve appeared stained a uniform red."

Michel concludes from his investigations that the progress of the degeneration of one optic nerve remains localized to the opposite tract. Cases 1 and 2 indicate this clearly. The varying involvement of the fibres of the opposite tract in both cases cannot be denied ; but whether the age of the patient or the nature of the ocular disease influenced this, is a question. Case 3 is more involved. It, however, speaks against the idea of a direct bundle, both in the sense of Hannover (lateral bundle) and in the sense of Gudden (a bundle lying laterally in the tract, and medially in the nerve), since the degree of atrophy is least marked in the ventral portion of the tract on the side of the enucleated eye.

"Further, as the degree of atrophy in the nerve of the eye not enucleated is less than in the tract of the opposite side, it would seem that the general picture was to be explained as a combination of an ascending atrophy of the left nerve with a descending atrophy of the left tract."

Michel looked for the cause of this uniform degeneration of the nerve corresponding to the eye not enucleated, and of the opposite tract, in the cystoid formations of the brain.

However this may be, it is certain that the case is not a simple, uncomplicated one, and it cannot, therefore, be considered either convincing or disproving. This may be said of case 4 also, in which no degenerated fibres were found in either tract. The cause of this may have lain in the method of staining, as these preparations were unfortunately stained with carmine, and this is insufficient to demonstrate the presence of partially degenerated medullated nerve fibres, especially in transverse sections.

There remain but two of Michel's cases in which the human chiasm was examined, which excite our interest, as *apparently* speaking against the idea of a semi-decussation in man. While even to-day it may rightly be objected to the statement of Gegenbaur's, cited above, that the direct bundle has not been anatomically demonstrated, we may also say with Mauthner: "The examination of preparations of the human chiasm with atrophy of one nerve teach indisputably that a semi-decussation takes place in the chiasm." Michel's cases 1 and 2 seemed destined, however, to weaken the anatomical proof of the semi-decussation. They would seem so, since they are the first pathological preparations of the chiasm which have been treated by a method which has long been considered as especially adapted for distinguishing normal medullated nerve fibres from degenerated ones. I hinted, however, at the Ophthalmological Society in August, 1887, and declared formally at the International Ophthalmological Congress in August, 1888, that these cases cannot weaken the theory of a semi-decussation, much less prove the total crossing. Before going further I would speak in some detail of my preliminary contribution, and of the preparations which I demonstrated at the two meetings mentioned.

I have already mentioned how in studying the development of the medullary sheaths in the embryonic chiasm, a method suggested itself to me which seemed in the highest degree adapted for following the course of the fibres by means of the development of their medullary sheaths. Weigert's method carefully carried out is the only known method by which healthy medullary substance can be made visible through the characteristic dark color. The difficult part of the process is the decolorizing.

In sections stained after Weigert's method the blood corpuscles also become more or less black, and they remain stained unless the action of the differentiating fluid is too long continued. If the differentiating fluid is allowed to act a somewhat longer time than is required for the unmedullated tissue to assume a yellow color, the blood corpuscles appear of a light-yellow color, like the remaining tissues. If the fluid acts still longer, the medullated fibres lose their color gradually and appear yellow, but of a darker tone than the blood and the other tissues. It is never found that the blood corpuscles remain black after the medullated nerve fibres have lost their color. The chiasm in the adult is only slightly vascular, but in the embryo and in young infants it is excessively vascular. In the tract, chiasm, and nerve, but especially in the gray matter filling the posterior angle of the chiasm, there are numerous microscopic and a considerable number of macroscopic vessels present, which in the young chiasm are distended with blood.

This fact makes it possible to be certain that all the medullary substance present is stained. In the process one need only watch the sections of the larger vessels and leave the specimen in the fluid as long as these vessels remain visible as dark points or stripes. This makes it certain that all the medullary substance is stained. Sections stained in this manner often show small dark precipitates, but since these merely lie on the tissue and are not chemically combined with it, they are easily washed off with distilled water. The specimens, as said before, may remain any length of time in water, but no longer in alcohol than is required for the dehydration. Thus carried out, the method never fails.

Chiasms of the twenty-eighth to the thirty-first week of embryonic life cannot be used for studying the course of the fibres, since the formation of medullary substance is not extensive and is limited to a few points. Chiasms of the thirty-second to the thirty-third week throw considerable light on the question.

If we look through the sections of the lower portion of the chiasm, the isolated medullated fibre bundles are seen to lie mostly parallel in the tract, but as they pass toward the chiasm they lie further apart and intercross under considerable angles. This crossing of the individual bundles is most marked where the nerve and tract pass into the chiasm. At the anterior angle of the chiasm, only such bundles are found as cross under very obtuse angles or possibly not at all. No fibres can be seen to change their direction and pass through the chiasm to the nerve of the same side. The picture is that of a total crossing of the medullated fibres. But in sections above the middle of the chiasm, at first a few, and going further upward a considerable number of fibres are seen which pass to the nerve of the same side. One is convinced that these are direct fibres, since fibres are seen which cross these and those previously described as crossing in a large arc, nearly at right angles.

In thus looking through the serial sections and following the portions of fibres showing a medullary sheath and varicosities, a considerable service is rendered by the light shining bundles of unmedullated fibres in which these medullated portions run; they make it possible to determine the direction of these, while it is, of course, impossible to follow an individual axis-cylinder accurately. It has not been possible, as I stated in my former communication, to follow the unbroken course of a medullated fibre from the tract into either nerve in specimens of this age and of the next older.

I advanced the opinion at that time, that this might be due to the fact that the individual fibres in a bundle interlace, and that therefore a section as thin as 10 μ could hardly strike a single fibre in its entire course.

But he who will take the trouble to make thin serial sections of the chiasm at this stage in its development, and study them section for section, following fibre after fibre, can come to no other conclusion than that **there are fibres in the upper half of the human chiasm which pass directly from one tract to the nerve of the same side.**

The straw mat or basket-like interweaving of the fibre bundles, first correctly described by Michel, can be very well seen in the parts of the chiasm near the nerves in those preparations in which the fibres are but partly medullated. It is seen at once in these incompletely developed chiasms that the fibre groups are so intimately interwoven in this characteristic manner, that a compact isolated course of the fibres, at least in the anterior part of the chiasm and in the nerves, cannot be spoken of. The three sorts of fibres are, certainly, at the points mentioned, interwoven in this basket fashion. In the tract, however, the medullated fibres and the axis-cylinders assume a more constant direction, and only the adjoining bundles cross each other at obtuse angles.

The study of chiasms up to those from infants of the third and fourth week merely confirmed the facts which have been stated. In these chiasms also, the direct fibres could be traced with certainty only in the upper half. In the lower half of the chiasm I have never been able to discover fibres having this course, but here all the fibres have the course which Michel has described for the fibres of the entire chiasm.

What may be said with reference to the relative numbers of crossed and direct fibres? The fact that the lower half of the chiasm contains only fibres which cross, and that the upper half contains both sorts mixed together, warrants us in assuming that **the number of crossed fibres is considerably greater than the number of direct ones.** The question, finally, as to whether the fibres run in a compact bundle, must, after a consideration of the foregoing observations, be answered in the negative.

The existence and the course of the two posterior commissures, Meynert's and Gudden's, has long been clear. Michel even has recognized them. Gudden's commissure lies in the chiasm and is intimately blended with it. The fibres form a narrow bundle, which may be said to bound the inner surfaces of the tracts and the posterior angle of the chiasm. The fibres of Meynert's commissure are of much greater calibre than any medullated fibres in the chiasm, and they lie outside the chiasm in the substantia cinerea of the tuber. Between this and Gudden's commissure, every section which includes both, shows a narrow stripe of gray matter between them.

Finally, I found in the middle of the chiasm, near Meynert's commissure, but clearly separated from this by gray matter, a varying number of small isolated bundles, which consisted of a few loosely joined fibres, and which lay scattered mostly in an oblique direction, back in the substance of the tuber cinereum. I can say nothing as to their origin or their further course. I had the impression, however, that they were in communication with the ganglionic groups which lie scattered through the substance of the tuber cinereum, although I was not able to find a direct connection of any such fibre with a ganglion cell. These are no doubt the same fibres which Gudden described (*Gr. Arch.*, vol. xxv., 1, p. 10), and which he was unable to find connected with particular ganglionic groups.

A word now in regard to the so-called Hannover-Erdl anterior commissure. In the beginning of my studies I believed that I found this in my preparations, but a more careful study of partly medullated sections showed me where I had been mistaken. The fibres which seem to pass unbroken from the inner side of one nerve through the anterior angle of the chiasm to the inner side of the other nerve, and so form a commissure, are only those crossed fibres which meeting the fibres of the opposite side exactly in the middle line of the anterior angle of the chiasm, cross nearly at a right angle and thus simulate commissural fibres. In this point I agree with Michel. The commissura ansata of Hannover did not come within the range of my investigations, since the surface of the chiasm was not studied. We may assume, however, that these fibres, if they are indeed nerve fibres, stand in no connection with the chiasm.

It is a welcome duty to mention another work of great importance, recently published in vol. lx. "Denkschr. der Naturwissensch. Classe der Kaiserl. Akad. der Wissensch. in Wien." This does not deal directly with the chiasm of man, but with that of the higher mammalia. A new method of staining employed clears up many unsettled questions.

The authors, J. Singer and E. Münzer,[33] have slightly modified a method described by Marchi and Algeri in 1887. When parts of nerve or brain which have been hardened in Müller's fluid are placed for five to eight days in a mixture of Müller's fluid and 1 per cent. osmic acid (2: 1), the degenerated and partially degenerated nerve fibres are seen under the microscope to be stained black, while the normal medullary sheaths are merely stained brown. In this manner a total crossing was found to exist in the dove, owl, mouse, and guinea-pig; while the rabbit, dog, and cat showed a partial crossing; the direct portion of the tract did not appear, however, in the form of a distinct bundle, but fibres belonging to it were found scattered over the entire transverse section of the tract. It was proven for the rabbit, in which the number of direct fibres is fewest, that the scattered direct fibres are found only in the upper sections of the chiasm and tract, just as we have described it in man.

This is apparently in direct contradiction with what Gudden and his pupils found in the rabbit's chiasm, viz., the compact course of the direct bundle. The authors have shown this contradiction to be only apparent, and due to the method employed. Gudden's method was to remove one eye of a new-born animal, after which the nerve and the portion of the tract belonging to it do not develop, and a transparent loose connective-tissue represents all that remains of the organ in question. If now, the rabbit has an uncrossed portion in the tract, which is made up of scattered fibres, as we have shown by Marchi's method, the employment of Gudden's method must give the following result: The scattered nerve fibres of the direct portion of the tract will be pressed together in the empty space left in consequence of the atrophy in the crossed tract, which latter does not develop after the enucleation of the eye, and thus simulate, to some extent, the existence of a separate isolated direct bundle. Similar secondary displacements have been repeatedly described by Gudden and his pupils in organs treated by his method, and this has been called by Mayser, "topical compensation." Indisputable proof of this hypothesis is furnished by Singer and Münzer. In a number of new-born rabbits one eye was enucleated directly after birth, the second four or five weeks later, and three weeks after this the animal killed and the chiasm examined by Marchi's method. The microscopic examination of the horizontal sections showed that the experiment had been successful.

The right nerve consisted of a narrow band of connective tissue, staining pale-brown by Marchi's method, without a trace of nerve fibres; the left nerve appeared intensely black, as well as the right tract. In the position of the left tract was seen the broad, well-developed, normal, and therefore pale-brown commissure of Gudden, on which lay a very narrow isolated bundle of fibres stained intensely black, and this bundle partly mingled its fibres with that bundle which in the left lateral angle of the chiasm separates from the degenerated optic nerve to pass to the tract of the same side. Similar experiments made on cats were followed by corresponding results. Singer and Münzer, therefore, regard the partial crossing as proven for the rabbit, dog, and cat, and the apparent contradiction of Gudden's statement in regard to the course of the direct fibres as satisfactorily explained. They say further: "The results of our investigations compel us to consider the conclusions expressed in Michel's monograph as incorrect. It would seem to us that the exclusive employment of Weigert's method was the cause of the error, since this method is not adapted for the examination of scattered degenerated fibres. It may be that refinements in technique may possibly enable us to get good results with this method. The reason why the stain is inadequate for this purpose is that the bits of medullary substance arising from Waller's degeneration stain in the same manner as the normal medullary sheaths, so that the degenerated fibres are not sharply enough distinguished from the normal. Had there been a compact separate bundle in the tract of the animals which Michel studied, Weigert's method would have revealed its atrophy ; the degeneration of the scattered fibres which our experiments have shown to exist, must have escaped him." It must certainly have escaped him in man.

My investigations have shown that the direct fibres in man do not lie so compactly together as was formerly supposed. In every section through the upper half of the chiasm, I found them mixed with the crossed fibres. The explanation of Michel's error, which Singer and Münzer have suggested, may be extended to the human chiasm.

I would willingly grant that Weigert's method is responsible for his error; still, I must hold to my previous opinion, and continue to believe that, if Michel had carried out the method of staining properly, and had examined a series of sections of the entire chiasm, some of the sections of the upper half must have shown Waller's degeneration or complete atrophy of some of the fibres. The important point in the technique is to wash the decolorized sections for hours in water, to which a few drops of lithium carbonate solution have been added.

Several years ago I had an opportunity, through the kindness of Professor Weigert, of examining in this way a chiasm with unilateral optic-nerve atrophy, where one eye had been lost in early youth. The sections of the lower half of the chiasm (plate x., fig. 1) presented exactly the picture which Michel has described for the entire chiasm.

The sections of the upper half of the chiasm showed a very different picture (plate x., fig. 2). In this part of the chiasm the nerve-fibres show Waller's degeneration in both tracts, and in many sections very distinctly in the uncrossed tract. In the uncrossed tract, besides fibres with normal sheaths, a number are seen whose medullary substance has in part disappeared, and which is in part in isolated irregularly-shaped bits. Many sections show, toward the inner side of the tract, a quantity of almost totally degenerated medullated fibres, with bits of medullary substance here and there (plate x., fig. 2*a*). The opposite tract shows many fibres equally atrophic. The nerve corresponding to the enucleated eye was completely atrophic, the other entirely normal. Why there are fibres in both tracts, showing various grades of atrophy, I am unable to say. The examination of this unilaterally atrophic chiasm, however, fully confirms my embryological proof of the semi-decussation, and shows also that Weigert's method is sufficient to demonstrate the direct fibres, provided that it be carried out with the necessary precautions, and that an unprejudiced observer examine every section in a complete series, following each bundle and each fibre.

I would here express my thanks to my honored instructor, Otto Becker, for his assistance in this investigation.

Explanation of the Figures.

FIG. 1.—Horizontal section from the lower (basal) half of the chiasm of a man with atrophy of the left optic nerve. Eye lost in early youth.

N. o. s. = Left optic nerve (completely atrophic).
N. o. d. = Right " " (normal).
T. o. s. = Left tract (normal).
T. o. d. = Right " (atrophic for the most part).
C. M. = Meynert's commissure.
G. c. = Gudden's "

FIG. 2.—Horizontal section from the upper (dorsal) half of the same chiasm.

N. o. s. = Left optic nerve (completely atrophic).
N. o. d. = Right " " (normal).
T. o. s. = Left tract (scattered, completely and partially atrophic fibres).
T. o. d. = Right tract (normal, excepting a few scattered partially atrophic fibres).
C. M. = Meynert's commissure.
C. G. = Gudden's "

FIG. 2. *a.*—A portion of the left tract more highly magnified.

a. Completely and partially atrophic uncrossed medullated fibres.
b. Normal varicose nerve fibres mixed with atrophic fibres.

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THE ACTION OF PRISMOSPHERES AND DECENTRED LENSES.

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(With five figures.)

PRISMS or prismatic combinations are frequently ordered for the relief of muscular defects of the eye, but they are by no means always successful. This is due partly to the great difficulty that is found in exactly determining the relative strength of the ocular muscles, but partly also, I believe, to a want of recognition of their precise action. The text-books to which I have referred for information on this point assume explicitly or implicitly that with a given prism the effect produced on the deviation of the eye is the same, whether it be combined with a spherical lens or not.[1] This is, however, very far from true.

We may examine the action of prisms and their combinations from two points of view, according to the purpose for which they are used. In the consulting-room they are chiefly used to effect an alteration in the position of the eyes, or rather in the direction of their fixation lines, but they are generally prescribed and worn to change the apparent site of objects, when binocular fixation[2] cannot be maintained without their aid. Obviously the angular displacement is the same in either case.

The action of plane prisms, when spherical lenses are not combined with them, is simple enough. If we denote the angle of deviation which the prism produces on a ray of light traversing it in the position of minimum deviation by Δ, the deviation (χ) of the fixation line is equal to this angle Δ, as the accompanying diagram indicates. This is an illustration of the advantage gained by designating prisms by their deviation : it is to be hoped that before long this practice will become universal.

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[1] In a small book by Mr. E. Maddox, "On the Clinical Use of Prisms," the difference is pointed out, but no formulæ are given for determining the extent of this difference.

[2] For practical purposes it may be assumed that the visual line coincides with the fixation line—*i. e.*, that joining the centre of rotation with the point fixed, unless the object of observation be very close to the eyes.

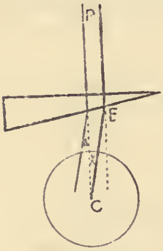

FIG 1

In order to simplify the diagram a rectangular prism, which is not in the position of min. dev., has been drawn ; c is the centre of rotation of the globe. The fixation line of an eye directed towards a distant object at P assumes the direction of the line C E after the interposition of the prism. Obviously angle χ = angle Δ.

When, however, a spherical lens is also placed in front of the eye in the normal position, it will be found that with but one exception the angle through which the eye rotates is no longer equal to the angle of deviation of the prism. *Vide* fig. 2.

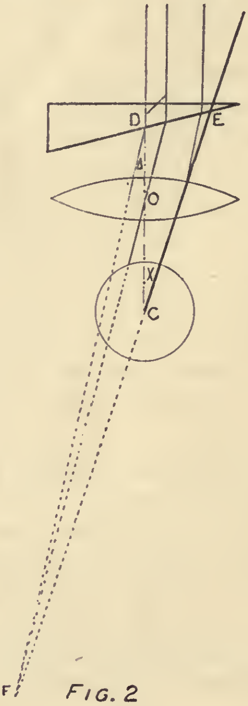

FIG. 2

Let F be the principal focus of the convex spherical lens O. Parallel rays, after traversing the prism, will be deflected through the angle Δ; on encountering the spherical lens they will be rendered convergent, intersecting at a point F on its secondary axis O F. C O is the primary direction of the fixation line. On placing this combination in front of the eye it will rotate through an angle χ, so that its fixation line now takes up the position C E. In the triangle D F C the exterior angle D C E is greater than the interior and opposite angle F D C $\therefore$ χ is greater than Δ.

If the spherical lens be concave χ is always less than Δ. A similar result is obtained when the lens is convex if the object of fixation be within its focal distance; should, however, the object be situated at the first principal focus in front of the eye, the angle χ is equal to the angle Δ. The effect of a plane prism with the object at infinite distance may be regarded as a special instance of this law.

A glance at fig. 3 shows that prismospheres are equivalent to decentred lenses and how prismatic combinations may be expressed in terms of decentred lenses.

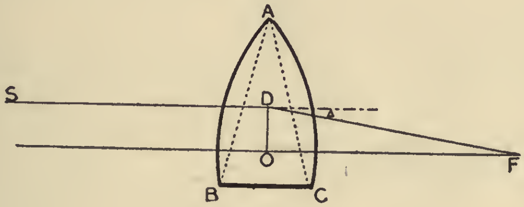

Fig. 3

A B C may be regarded as a decentred lens or as a combination of the prism (marked on dotted lines) with a spherical lens. In the prismosphere A B C, O is the optical centre of the equivalent lens, D is its geometrical centre, O D being the decentration. An incident ray, S D, traversing the point D will be deflected towards the point F, the principal focus of the lens; the angle of its deviation Δ is obviously equal to the angle D F O, $\therefore \tan\Delta = \frac{O\,D}{O\,F}$ or $\frac{d}{f}$ where d represents the decentration and f the focal distance of the lens; D represents the power of the lens in dioptries; $\frac{d}{f}$ becomes $\frac{d\,D}{1000}$. When Δ is small no great error is introduced by considering it proportional to $\tan\Delta$.

Then $\Delta = d\,D \tan^{-1}.001 = d\,D \times 3'.4375$[1]—*e. g.*, a decentration of a + 12 D lens to extent of 6.7 *mm* will be equivalent to a prism of 4° 36′ d. (= $6.7 \times 12 \times 3'.4375$).

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[1] Even when $d\,D = 120$ an error of less than $2'$ is introduced.

It will be convenient in studying the action of prismospheres to regard them as decentred lenses and to examine their effect on the direction of the ocular fixation lines. A general formula can then be found by a very simple geometrical method.

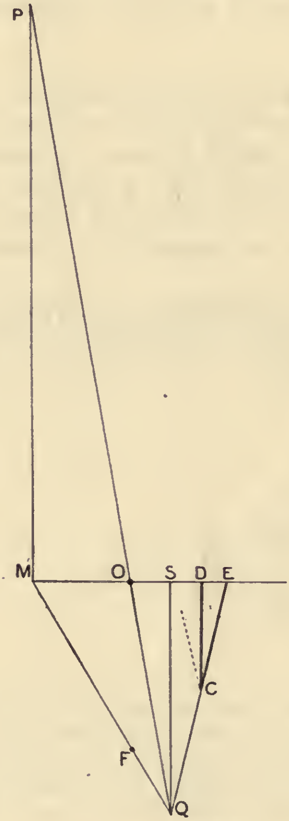

FIG. 4

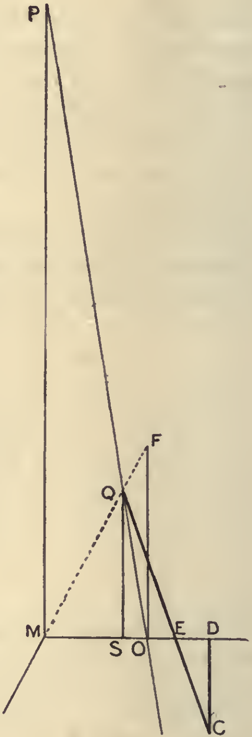

FIG 5

Let the case of a convex lens be taken first, F being its 2d principal focus, O its optical centre, D its geometrical centre, its decentration therefore is O D, which may be denoted by the symbol d, and may be given the positive sign when the displacement is inwards. Let P be a point situated at a distance M P or p from the plane of the glass, and let Q be its image at distance Q S or q from the glass. Let C be the centre of rotation of the eye at distance D C or k behind the lens. Join QC and produce it to E. Then C E represents the direction of the fixation line for the point P. In fact an eye in order to see P must rotate through the angle P C E after the decentred lens has been interposed. In practice it is convenient to measure the deviation of the eye from its primary position C D. Let χ (D C E) denote this angular deviation and let it be considered positive when it induces divergence of the visual lines.

Now $\frac{OS}{SQ} = \frac{MO}{MP}$. ∴ $OS = \frac{q}{p}.MO = \frac{q}{p}(MD - d)$, but $SD = OD - OS = d - \frac{q}{p}(MD - d)$.

Again $\frac{SQ}{DC} = \frac{SD + DE}{DE}$, or $\frac{q}{k} = \frac{d - \frac{q}{p}(MD - d) + DE}{DE}$.

$$\therefore \tan\chi \text{ or } \frac{DE}{k} = \frac{d - \frac{q}{p}(MD - d)}{q - k} = \frac{d.\frac{p + q}{p} - \frac{q}{p}.MD}{q - k}$$

Now if the incident pencil from P do not strike the lens too obliquely the formula $\frac{1}{f} = \frac{1}{p} + \frac{1}{q} = \frac{p + q}{pq}$ holds good.

$$\therefore \tan\chi = \frac{d\frac{q}{f} - \frac{q}{p}.MD.}{q - k}$$

On examination of this formula the following relations are obtained :

(i) When p is infinite $q = f$, $\tan\chi = \frac{d}{f - k}$

(ii) When object immediately in front of eye $MD = 0$, $\tan\chi = \frac{d\frac{q}{f}}{q - k}$

(iii) On eliminating q from the expression $\frac{d\frac{q}{f} - \frac{q}{p}MD}{q - k}$, $\tan\chi = \frac{dp - fMD}{fp - k(p - f)}$ since $q = \frac{fp}{p - f}$

(a) When the object is immediately in front of the eye, its distance p being equal to f, $\tan\chi = \frac{d}{f}$ *i. e.*, $= \tan\triangle$ where $\triangle$ is the deviation produced by the virtual prism.

(β) When p is less than f $\tan\chi$ and therefore χ becomes negative.

(iv) When lens concave, f is negative, thus $\tan\chi = -\frac{dp + fMD}{fp + k(p + f)}$, but if p be infinite, $\tan\chi = -\frac{d}{f + k}$

∴ If the decentration is positive (*i. e.*, inwards) χ is negative and *vice versa.*

These results may be obtained geometrically, and to ensure recognition of the general applicability of the formula a figure for the case of a concave lens is appended. (Fig. 5.)

A concave lens having its focus at F is decentred to the extent indicated by O D. C represents the centre of rotation of the eye, Q is the virtual image of the point P, C E Q gives the direction of the fixation line. The decentration in this case causes convergence. ∴ χ is negative.

As before $OS = \frac{SQ}{MP}$. $MO = \frac{q}{p}(MD - d)$, but $SD = OD + OS = d + \frac{q}{p}(MD - d)$.

Now $\frac{SQ}{DC} = \frac{SD - DE}{DE}$, or $\frac{q}{k} = \frac{d + \frac{q}{p}(MD - d) - DE}{DE}$

$\therefore \frac{DE}{k}$ or $\tan(-\chi) = \frac{d + \frac{q}{p}(MD - d)}{q + k}$ but $\frac{1}{p} - \frac{1}{q} = -\frac{1}{f} \therefore q = \frac{fp}{p + f}$

Substituting this expression for q, $\tan(-\chi) = \frac{dp + f.MD}{fp + k(p + f)}$

This expression enables one to determine the degree of ocular deviation produced by a given decentred lens, and conversely one can find out to what extent a given lens should be decentred in order to induce in the fixation lines a given direction.

If the lens be convex, $d = \frac{fMD \pm \tan\chi(fp - k\overline{p - f})}{p}$, the +^{ve} sign being used if divergence is produced, the −^{ve} if convergence.

If the lens be concave, $d = -\frac{f.MD \pm \tan\chi(fp + k\overline{p + f})}{p}$. In this case obviously the decentration must in all cases be negative or outwards, in order to produce divergence.

If the fixation lines are to maintain parallelism $\tan\chi$ becomes 0 and $d = \pm\frac{f.MD}{p}$.

It will be seen that the increments of *d* will be proportional to the increments of tan χ; if for instance we were to tabulate the values of *d* for an arithmetic series of values of tan χ, it would be found that they also formed an arithmetic series. In other words, the tabular differences would be constant.[1]

The formulæ contain four interdependent variables and two constants. Since glasses should be placed in the first principal focal plane of the eye, which is situated 27.4751 *mm* from the centre of rotation of the globe, this value should be assigned to *k*.[2] The distance between the ocular centres has been assumed to be 64 *mm*, this being its usual value in adults; if the object be in the middle line, M D may be replaced by 32 *mm*. I have drawn up a series of tables for reference in this way by assigning their respective values to the variables in these formulæ, the distance of the object being reckoned from the centre of rotation of the globe. The tabular differences corresponding to tan 1° and tan 1 ma have been calculated to three places of decimals.[1]

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[1] In mathematical language d is an integral function of tan χ of the first degree, its increment therefore is proportional to the increment of its argument (tan χ).

[2] The value of k is deduced from the dimensions given in Landolt's article in the *Traité Complet d'Ophthalmologie*, vol. iii., pp. 83, 110.

Table I. is to be used when adaptation of the eyes for distant vision is required, the upper half giving figures for convex lenses, the lower for concave. Suppose a hypermetrope of 8 D present himself, and when his accommodation is relaxed it is found that his minimum convergence is 1 ma—*i. e.*, that his fixation lines intersect at a point 1 metre from him. Since the lens is convex and divergence is required, the decentration must be inwards, and a reference to the table at once gives its amount 3.121 *mm*. Had the patient been myopic to the extent of 8 D with min. convergence of 2°, he would require — 8 D glasses decentred outwards 2 χ 2.661 *mm* or 5.322 *mm*.

In Tables II. and III. the object of observation is supposed to be at $\frac{1}{3}$ *m* from the centre of rotation of the globe—*i. e.*, 305.858 *mm* from the plane of the glasses. Having found what degree of convergence can be maintained without fatigue relatively to the accommodation exerted, it is a simple matter to correct the convergence defect. A patient requiring convex lenses of + 12 D for reading (at $\frac{1}{3}$ *m*) can only maintain convergence for a distance of 1 *m* (1 ma). On reference to Table II. it is at once seen that his glasses must be decentred inwards 6.7 *mm*. Were his spectacles normally centred, more than 4 ma of convergence would be required in order to obtain binocular vision of an object $\frac{1}{3}$ *m* from him. Table III. shows that a myope using —12 D for reading must have them decentred outwards 8.7 *mm*, if he have no power of positive convergence. Binocular vision of the object can then be maintained with parallel visual lines.

The prismatic equivalents have been printed in smaller type just below the numbers which indicate the amount of decentration for the convenience of those who prefer to prescribe such corrections in terms of prisms rather than as decentred lenses. Thus in the last instance the — 12 D spectacles, decentred outwards 8.7 *mm*, may be prescribed, or, what comes to the same thing, prisms of 5° 58′ d, with their refracting surfaces ground concave to the extent of – 12 D, may be ordered to be mounted in the frames with their edges directed towards the temples. Though in the examples given only errors of convergence have been taken, it is obvious that the use of the tables is not confined to the relief of such defects, but that Table I. at any rate is equally applicable in cases of hyperphoria.

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[1] Constancy of the tabular differences for angles may be assumed within the limits for which these tables are used. Thus, 10 tan 7° = .174551 = tan 9° — 54'.0767. In this case, therefore, an error of less than 6' is introduced by assuming that angles are simply proportional to their tangents.

Enough has now been said to explain the way in which prismatic combinations may be correctly prescribed when the muscular object is known. It is hoped that these tables may also prove of service in diagnosing more precisely than has hitherto been customary the relative strength of the ocular muscles.

In examining the binocular function, it is important that the retinal images should be defined, in order to stimulate the desire for fusion. Hence in all cases in which errors of refraction exist, these should be first corrected by appropriate lenses. Thus, in determining the (absolute) minimum of convergence, the patient should first be provided with glasses, which enable him to define some distant object without exerting his accommodation. While his attention is now concentrated upon it, abducting prisms are placed before the glasses; the strongest prism compatible with single vision enables one to discover the minimum of convergence. It is sometimes found that hypermetropes can overcome stronger prisms without the correcting glasses. As an example of this, I may take a case I examined a few days ago. The patient had compound hypermetropic astigmatism:

R. H^m + 4 D sph. $\overset{\frown}{\smile}$ + 1.5 D cyl. axis | 90°
L. H^m + 2 D sph. $\overset{\frown}{\smile}$ + 1 D cyl. axis | 90°

With these lenses duly adjusted, the strongest abducting prism held before the right eye that could be overcome was found to be one of 6° d, but without these glasses binocular vision could be easily maintained with a prism of 6° 30′ d, and momentarily attained with one of 7° d. At first sight it would appear that his power of divergence was greater when he was exercising his accommodation than when it was at rest, but a little further consideration on what has gone before will show that this is not the case. A 6° d prism held before the sphero-cylinder + 4 D Sph. ⊃ + 1.5 D cyl. ax | 90°, will cause an angular deviation of about 7° 3′ in the direction of the fixation line.[1]

The same prism (6° d), when placed before the left eye and its correcting glass, produces only a deviation of 6° 32′. In the case in question, the patient said the prism seemed to strain his left eye less than his right. In many cases, probably, the diverging power will be found to be considerably greater when the accommodation is completely relaxed, and the refraction of the eye adjusted for distance by means of glasses, than when such precautions are not taken. If the ametropia be of sufficiently high degree, the power of divergence may be estimated by altering the position of the glasses, separating them if concave, approximating them if convex. When the decentration and the dioptric strength of the glass is known, Table I. gives the angle of divergence or minimum convergence.

The exact determination of the absolute maximum of convergence is a more difficult matter. The old finger test is a simple, rough method, which has been modified in various ways. Landolt's plan is merely a modification of the same principle, the object of fixation being a line of light, which is gradually brought close to the patient; as soon as the line appears double, the distance from the eye to the object is measured.

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[1] The power of the horizontal axis of the sphero-cylinder is + 5.5 D, and from Table I. it will be seen that a 6° d prism must cause a deviation of rather less than 7° 17′. If greater accuracy be demanded, the formulæ given above may be used. $d = f \tan \triangle = \frac{1000}{5.5} . \tan 6° = \frac{105.1042}{5.5} =$ 19.11, the effect on the direction of the fixation line is therefore $\tan^{-1} \frac{19.11}{f - k} =$

$$\tan^{-1} \frac{19.11}{181.81 - 27.4751} = 7° \; 3' \text{ approximately.}$$

Much can be said in favor of this method; its simplicity the readiness with which the test can be applied, and the short time the examination occupies, recommend it strongly both to doctor and patient. There are, however, some serious objections to its use; I believe it indeed to be quite unreliable in cases of hypermetropia. When the point of fixation falls within the binocular near point of accommodation, the retinal images are ill-defined, and hence can hardly stimulate the desire for fusion efficiently. If the power of convergence be great, a small error in measurement will seriously impair the accuracy of the result. The difference between 1 ma and 2 ma corresponds to a distance of 50 *cm*, but that between 10 ma and 11 ma is represented by a distance of less than 1 *cm*. Precise measurement is difficult, as the reckoning should be made from a point midway between the ocular centres.

In default of a better, I would tentatively suggest the following procedure; I cannot as yet speak very confidently about it, as I have not yet tested its accuracy in a sufficient number of cases. After the examination of the refraction and the accommodation of the eyes, glasses should be given, such that the near point of accommodation (pa) becomes $\frac{1}{3}$ *m*. The strongest adducting prisms compatible with single vision must now be found. The position of the near point of convergence (pc) can then be determined, either by the help of the tables or by means of a simple calculation.

For instance, a hypermetrope of + 7 D, with accommodation equal to + 4 D, will require a + 6 D lens to bring pa to $\frac{1}{3}$ *m*. Convex glasses of this strength must now be given, and accurately centred as if for distant vision. The power of convergence must now be tested, either by separating the lenses or by prisms. Suppose that in this case the limit of convergence is reached when each eye is furnished with a 14° d prism. On referring to Table II. it will be seen that an adducting prism of 47′, combined with a + 6 D lens, entails a convergence of 4 ma when the object of fixation is at $\frac{1}{3}$ *m*. Now the addition of a prism of 1° 42′ increases the convergence by 1 ma: a prism of 14° — 47′ must therefore increase the convergence by $\frac{13^\circ\ 13'}{1^\circ\ 42'}$ ma, *i. e.*, 7.7 ma, if we assume that the angles are proportional to their tangents. Hence the total convergence is 4 + 7.7 ma, or 11.7 ma approximately.

Greater accuracy is obtained when the value of the prism is expressed in terms of decentration of the lens. The 14° d prism is equivalent to a decentration outwards in a + 6 D lens of 41.554 *mm* ($\frac{1000 \tan 14^\circ}{6}$ *mm*). On reference to Table II. it will be seen that a + 6 D lens must be decentred 17.4372 *mm* inward in order to annul convergence, and that 1 ma corresponds to a decentration of 4.933 *mm*. Hence the convergence is represented by $\frac{41.554 + 17.437}{4.933}$, or 11.958 ma. The near point of convergence is therefore 8.36 *cm*, corresponding to an angular deviation of 20° 56′.

It is only by precision in diagnosis and accuracy in the prescription of correcting glasses that satisfactory results can be obtained in the treatment of muscular asthenopia.

In order to avoid the want of definition due to the chromatic dispersion of strong prisms, I make this examination in the dark room, using monochromatic light to illuminate the object of fixation, *e. g.*, a spirit lamp which volatilizes some sodium carbonate.

In cases in which pa nearly coincides with pc (*e. g.*, myopia) Landolt's dynamometer gives accurate results and is very convenient.

Prisms are sometimes chosen at random, and ordered to be worn in the hope that they will relieve certain symptoms; it is not surprising that failure attends such unscientific treatment. Some discredit has fallen upon them, and many surgeons have abandoned their use, advocating operative procedures in their stead. It is with no intention of condemning such operations that I venture to offer a plea for a *fair* trial of prismospheres. The cure of muscular asthenopia, with all the varied and distressing symptoms it occasions in neurotic individuals, is as gratifying as it is difficult, but when the diagnosis is exact and the treatment scientific success is certain.

TABLE I.

				1 D	2 D	3 D	4 D	5 D	6 D	7 D	8 D	9 D	10 D	12 D	14 D	16 D	18 D	20 D
CONVEX.	Divergence decentration inwards. Convergence " outwards.	4 ma.	7°17′	124.5 7°6′	60.5 6°54′	39.1 6°42′	28.5 6°30′	22.1 6°18′	17.8 6°6′	14.7 5°54′	12.5 5°41′	10.7 5°30′	9.3 5°18′	7.1 4°54′	5.6 4°30′	4.5 4°6′	3.6 3°42′	2.9 3°18′
		3 ma.	5°29′	93.3 5°20′	45.3 5°11′	29.3 5°2′	21.3 4°53′	16.5 4°44′	13.3 4°35′	11.1 4°26′	9.3 4°17′	8.0 4°7′	6.9 3°59′	5.3 3°41′	4.2 3°23′	3.3 3°5′	2.7 2°47′	2.1 2°29′
		2 ma.	3°40′	62.2 3°33′	30.2 3°28′	19.6 3°21′	14.2 3°16′	11.0 3°9′	8.9 3°4′	7.4 2°57′	6.2 2°52′	5.3 3°45′	4.6 2°40′	3.6 2°27′	2.8 2°15′	2.2 2°3′	1.8 1°51′	1.4 1°39′
		1 ma.	1°50′	31.120 1°47′	15.121 1°44′	9.787 1°41′	7.121 1°38′	5.521 1°35′	4.454 1°32′	3.692 1°29′	3.121 1°26′	2.676 1°23′	2.321 1°20′	1.787 1°14′	1.406 1°8′	1.121 1°2′	.898 55′	.721 50′
			1° 0	16.975 58′	8.248 57′	5.339 55′	3.884 53′	3.011 52′	2.429 50′	2.014 48′	1.702 47′	1.460 45′	1.266 43′	.975 40′	.767 37′	.611 33′	.490 30′	.393 27′
CONCAVE.	Convergence decentration inwards. Divergence " outwards.		1°	17.935 1°2′	9.207 1°3′	6.298 1°5′	4.843 1°6′	3.970 1°8′	3.389 1°10′	2.973 1°12′	2.661 1°13′	2.419 1°15′	2.225 1°16′	1.934 1°20′	1.726 1°23′	1.570 1°26′	1.449 1°30′	1.352 1°33′
		1 ma.	1°50′	32.879 1°53′	16.879 1°56′	11.546 1°59′	8.879 2°2′	7.279 2°5′	6.212 2°8′	5.450 2°11′	4.879 2°14′	4.435 2°17′	4.079 2°20′	3.546 2°26′	3.165 2°32′	2.879 2°38′	2.657 2°44′	2.479 2°50′
		2 ma.	3°40′	65.75 3°45′	33.7 3°52′	23.1 3°57′	17.7 4°4′	14.5 4°10′	12.4 4°15′	10.9 4°22′	9.7 4°28′	8.8 4°34′	8.1 4°40′	7.1 4°52′	6.3 5°4′	5.7 5°16′	5.3 5°28′	4.9 5°36′
		3 ma.	5°29′	98.6 5°38′	50.6 5°47′	34.6 5°56′	26.6 6°5′	21.8 6°14′	18.6 6°23′	16.3 6°32′	14.6 6°41′	13.3 6°50′	12.2 9°59′	10.6 7°16′	9.5 7°24′	8.6 7°42′	7.9 8°10′	7.4 8°27′
		4 ma.	7°17′	131.5 7°29′	67.5 7°41′	46.2 7°53′	35.5 8°5′	29.1 8°17′	24.8 8°29′	21.8 8°41′	19.5 8°52′	17.7 9°4′	16.3 9°16′	14.2 9°39′	12.6 10°3′	11.5 10°26′	10.6 10°50′	9.9 11°12′

The object of observation is presumed to be at a distance of more than 6 metres from the patient.
The figures in larger type indicate the amount of decentration in millimetres.
The figures in smaller type represent the deviating power of the prisms whose action is equivalent to that of the decentration of the lenses.

TABLE II.—(Convex.)

			1 D	2 D	3 D	4 D	5 D	6 D	7 D	8 D	9 D	10 D	12 D	14 D	16 D	18 D	20 D
Diverging.	4 ma.	7°17′	240.6 13°32′	118.5 13°20′	77.8 13°8′	57.5 12°57′	45.3 12°46′	37.2 12°34′	31.3 12°22′	27.0 12°11′	23.6 11°59′	20.9 11°48′	16.8 11°26′	13.9 11°2′	11.7 10°47′	10.0 10°21′	8.7 9°51′
	3 ma.	5°29′	206.6 11°40′	101.9 11°31′	67.1 11°23′	49.7 11°14′	39.2 11°6′	32.2 10°57′	27.2 10°48′	23.5 10°39′	20.6 10°31′	18.3 10°22′	14.8 10°4′	12.3 9°47′	10.4 9°29′	8.9 9°11′	7.8 8°54′
	2 ma.	3°40′	172.6 9°47′	85.4 9°42′	56.3 9°36′	41.8 9°30′	33.1 9°24′	27.3 9°18′	23.1 9°12′	20.0 9°7′	17.6 9°1′	15.7 8°55′	12.8 8°43′	10.7 8°31′	9.1 8°17′	7.9 8°7′	6.9 7°56′
	1 ma.	1°50′	138·6 7°53′	68.8 7°50′	45.6 7°48′	34.0 7°45′	27.0 7°42′	22.3 7°39′	19.0 7°36′	16.5 7°33′	14.6 7°30′	13.1 7°27′	10.7 7°21′	9.1 7°15′	7.8 7°9′	6.8 7°3′	6.1 6°57′
		0	104.6236 5°58′	52.3118 5°58′	34.8745 5°58′	26.1559 5°58′	20.9247 5°58′	17.4372 5°58′	14.9462 5°58′	13.0779 5°58′	11.6248 5°58″	10.4623 5°58′	8.7186 5°58′	7.4731 5°58′	6.5389 5°58′	5.8124 5°58′	5.2312 5°58′
Converging.	1 ma.	1°50′	70.6 4°2′	35.7 4°5′	24.1 4°8′	18.3 4°11′	14.8 4°14′	12.5 4°18′	10.8 4°21′	9.6 4°23′	8.6 4°26′	7.8 4°29′	6.7 4°36′	5.8 4°41′	5.2 4°47′	4.7 4°53′	4.3 4°59′
	2 ma.	3°40′	36.6 2°6′	19.2 2°12′	13.4 2°18′	10.5 2°24′	8.7 2°30′	7.6 2°36′	6.7 2°42′	6.1 2°48′	5.6 2°54′	5.2 3°	4.6 3°12′	4.2 3°24′	3.9 3°36′	3.7 3°48′	3.5 4°
	3 ma.	5°29′	2.6 9′	2.6 18′	2.6 27′	2.6 36′	2.6 45′	2.6 54′	2.6 1°3′	2.6 1°13′	2.6 1°22′	2.6 1°31′	2.6 1°49′	2.6 2°7′	2.6 2°25′	2.6 2°43′	2.6 3°1′
	4 ma.	7°17′	−31.3 (1°48′)	−13.9 (1°36′)	−8.1 (1°24′)	−5.2 (1°12′)	−3.4 (59′)	−2.3 (47′)	−1.4 (35′)	−.84 (23′)	−.36 (11′)	.03 1′	.61 25′	1.02 49′	1.3 1°13′	1.58 1°37′	1.77 2°2′
Difference for 1 ma.			33.995 1°57′	16.558 1°54′	10.743 1°51′	7.839 1°48′	6.0957 1°45′	4.933 1°42′	4.1029 1°39′	3.4801 1°36′	2.9957 1°33′	2.6082 1°30′	2.0270 1°24′	1.6119 1°18′	1.3004 1°12′	1.0582 1°6′	.8645 59′
Difference for 1°			18.54 1°3′	9.032 1°2′	5.861 1°	4.276 59′	3.325 57′	2.691 55′	2.2380 53′	1.8983 52′	1.6341 50′	1.4227 49′	1.1057 46′	.8792 42′	.7093 39′	.5772 36′	.4716 32′

The object of observation is presented to be ⅓ metre from the centre of rotation of the globe.
The figures in larger type give the amount of decentration in millimetres.
—ᵛᵉ sign indicates decentration outwards; +ᵛᵉ sign decentration inwards.
The figures in smaller type represent the deviating power of the prisms whose action is equivalent to that of the decentration of the lenses. When enclosed in brackets the prisms are adducting in junction, and should be placed edges inwards.

TABLE III.—(CONCAVE.)

			−1 D	−2 D	−3 D	−4 D	−5 D	−6 D	−7 D	−8 D	−9 D	10 D	−12 D	−14 D	−16 D	−18 D	−20 D
DIVERGING.	4 ma.	7°17′	−247.6 13°55′	−125.6 14°6′	−84.9 14°17′	−64.5 14°28′	−52.3 14°40′	−44.2 14°51′	−38.4 15°2′	−34.0 15°14′	−30.6 15°25′	−27.9 15°36′	−23.8 15°59′	−20.9 16°21′	−18.8 16°43′	−17.1 17°5′	−15.7 17°27′
	3 ma.	5°29′	−211.9 11°58′	−107.2 12°7′	−72.4 12°15′	−54.9 12°24′	−44.5 12°32′	−37.5 12°41′	−32.5 12°49′	−28.8 12°58′	−25.9 13°7′	−23.5 13°16′	−20.1 13°33′	−17.6 13°50′	−15.7 14°7′	−14.3 14°24′	−13.1 14°41′
	2 ma.	3°40′	−176.1 9°59′	−88.9 10°5′	−59.9 10°11′	−45.3 10°17′	−36.6 10°23′	−30.8 10°29′	−26.6 10°35′	−23.5 10°40′	−21.1 10°46′	−19.2 10°52′	−16.3 11°4′	−14.2 11°15′	−12.6 11°27′	−11.4 11°39′	−10.5 11°50′
	1 ma.	1°50′	−140.4 7°59′	−70.6 8°2′	−47.4 8°5′	−35.7 8°8′	−28.8 8°11′	−24.1 8°14′	−20.8 8°17′	−18.3 8°20′	−16.4 8°23′	−14.8 8°26′	−12.5 8°32′	−10.8 8°38′	−9.6 8°44′	−8.6 8°50′	−7.8 8°56′
		0	−104.6236 5°58′	−52.3118 5°58′	−34.8745 5°58′	−26.1559 5°58′	−20.9247 5°58′	−17.4372 5°58′	−14.9462 5 °58′	−13.0779 5°58′	−11.6248 5°58′	−10.4623 5°58′	−8.7186 5°58′	−7.4731 5°58′	−6.539 5°58′	−5.8124 5°58′	−5.2312 5°58′
CONVERGING.	1 ma.	1°50′	−68.9 3°56′	−34.0 3°53	−22.4 3°50′	−16.5 3°47′	−13.1 3°44′	−10.7 3°41′	−9.1 3°38′	−7.8 3°35′	−6.9 3°32′	−6.1 3°29′	−4.9 3°23′	−4.1 3°17′	−3.5 3°11′	−3.0 3°5′	−2.6 2°59′
	2 ma.	3°40′	−33.1 1°54′	−15.7 1°48′	−9.8 1°42′	−6.9 1°36′	−5.2 1°30′	−4.0 1°24	−3.2 1°18′	−2.6 1°12′	−2.1 1°5′	−1.7 59′	−1.1 47′	−.73 35′	−.42 23′	−.18 11′	.01 (1′)
	3 ma.	5°29′	2.6 (9′)	2.6 (18′)	2.6 (27′)	−2.6 (36′)	2.6 (45′)	2.6 (54′)	2.6 (1°3′)	2.6 (1°12′)	2.6 (1°21′)	2.6 (1°31′)	2.6 (1°49′)	2.6 (2°7′)	2.6 (2°25′)	2.6 (2°43′)	2.6 (3°1′)
	4 ma.	7°17′	38.4 (2°12′)	20.9 (2°24′)	15.1 (2°36′)	12.2 (2°48)	10.5 (3°)	9.3 (3°12′)	8.5 (3°24′)	7.9 (3°36′)	7.4 (3°48′)	7.0 (4°)	6.4 (4°24′)	6.0 (4°48′)	5.7 (5°12′)	5.4 (5°36′)	5.2 (6°)
Difference for 1 ma.			35.754 2°3′	18.316 2°6′	12.504 2°9′	9.598 2°12′	7.854 2°15′	6.6916 2°18′	5.8613 2°21′	5.2385 2°24′	4.7541 2°27′	4.3667 2°30′	3.7854 2°36′	3.3702 2°42′	3.0588 2°48′	2.8167 2°54′	2.6229 3°
Difference for 1°			19.502 1°7′	10.088 1°9′	6.820 1°10′	5.235 1°12′	4.284 1°14′	3.650 1°15′	3.1971 1°17′	2.8574 1°18′	2.5932 1°20′	2.3819 1°22′	2.0648 1°25′	1.8383 1°28′	1.6685 1°32′	1.5364 1°35′	1.4307 1°38′

The object of observation is presumed to be $\frac{1}{3}$ metre from the centre of rotation of the globe.

The figures in larger type give the amount of decentration in millimetres.

$-^{ve}$ sign indicates decentration outwards; $+^{ve}$ sign decentration inwards.

The figures in smaller type represent the deviating power of the prisms whose action is equivalent to that of the decentration of the lenses. When enclosed in brackets the prisms are adducting in junction, and should be placed edges inwards.

THE RESULTS OF THE VARIOUS METHODS OF EXTRACTION OF CATARACT, ILLUSTRATED BY TWO HUNDRED AND SIX CASES.

By D. B. St. JOHN ROOSA, M.D., LL.D.

IT is quite possible, that the value of statistics of the operation for extraction of cataract has very much diminished of late years, since the publication of voluminous and interesting tables illustrating this subject; but I have thought that there would be a certain interest in an additional paper on the extraction of the cataractous lens, if it gave, as will the one that I have the honor to present, the statistics of one operator in a series of cases, many of which were observed for several years. I have hitherto refrained from any such publication, for the reason that I have not believed that what I had to say, was of sufficient importance to break in on the line of valuable contributions that have been made by many authorities in ophthalmic practice.

Now, however, that we know from thousands of reported cases, about what percentage in the hands of an average operator will result in useful vision, I believe even the report of a moderate number of consecutive cases, operated upon by different methods, yet by one operator, and followed up, in many instances, by years of observation, will have a certain interest, entitling it to be presented to my brother ophthalmologists. My paper will thus exhibit some of the vicissitudes of patients who have been operated upon for cataract. The cases are in no sense selected ones, for I have omitted none of which I have any adequate memoranda, and I have been in the habit of rejecting no cases as unfit for operation in which there was any chance of a good result. Connected, since its organization, with the Manhattan Eye and Ear Hospital, where Agnew, *facile princeps* as an operator, gave so many years of faithful and brilliant service, it is not to be wondered at, that, in common with his other colleagues in this institution, I have been glad to take the cases that did not demand him.

The report embraces all the cases that I have ever operated upon by extraction, with the exception of an uncertain number, probably twenty in all, operated upon at Burlington, Vermont, in the clinic of the university of that State, whose records have been destroyed or lost. The cases comprise such as I have operated upon at the Manhattan Eye and Ear Hospital, in the Burlington clinic just alluded to, and in my private practice.

I have now been operating for cataract since 1865. It will be seen that I began when Beer's knife was almost universally employed, and the ordinary flap without an iridectomy was made. I was taught the old-flap extraction at the hands of Schuft-Waldau, and Graefe, in Berlin, and Arlt, in Vienna. Since then have come Graefe's knife, and his operation with an iridectomy, Liebreich's operation and its imitations, the operations for the removal of the lens in its capsule, and finally the return to the simple flap operation with Graefe's knife and without an iridectomy.

It is true that many operators, especially in England, adhere to the concurrent iridectomy, but the general consensus seems to be in favor of what is termed simple extraction. My cases illustrate in a minor way, all these phases of changes in the professional mind upon this important subject.

They may have a little interest from that standpoint. Be that as it may, they certainly will be entitled to a slight consideration, at least, because they represent the experience of one operator through two decades of professional work. As I have already said, I have had the rare opportunity of observing some of these cases for years, and consequently of noting what is very little written about, so far as I have seen—the changes which occur to the eye without a lens.

They illustrate something also (especially in two families in each of which I have performed three operations)—what heredity may have to do with the formation of cataract.

The greatest change that has come upon this important operation, is the use of Koller's discovery of the anæsthetic property of cocaine. This may be said to have deprived the operation of extraction of nearly all its horrors, because we were always between Scylla and Charybdis before we had cocaine. There was the restlessness and the pain suffered by the patient, the liability to uncontrollable movement of the eye, if one operated without an anæsthetic, and the occasional deplorable consequences of the anæsthetic upon the healing, or the beginning of the healing, of the wound. Certainly one of the failures here reported may be ascribed to the struggling during incomplete anæsthesis, induced by the use of ether, although in the hands of an experienced person. And yet, in another case, nervous agitation produced by the operation without an anæsthetic caused such a shaking of the head immediately after it was finished, although the patient had nerved herself to its performance, as to cause entire loss of the eye. The earlier ones are those of an inexperienced operator, the later ones were performed when more skill in the technique and more judgment had been acquired by the years of practice.

I have never become a convert to the active local antiseptic measures that have been practised in many of the clinics, and which have had some advocates so earnest that they have almost denounced those as guilty of malpractice who failed to comply with the latest antiseptic precautions, as laid down by the latest writer on this subject. I do not underrate in any manner the magnitude of Lister's great work, the importance of the subject of absolute cleanliness, on which after all the whole thing turns, and I have become more and more careful as to the cleansing of the eye from all external irritants, the previous treatment of any conjunctival or lachrymal catarrh, the cleansing of the instruments, of the hands of the operator and those of his assistants; but I do not use carbolic acid or bichloride, or any of the special antiseptic fluids, but simply a boracic acid solution for cleansing the eye, hot water and soap for the instruments, dipping them in boiling hot water just before using, and zealously looking after the forceps with a magnifying glass. Some of the antiseptic enthusiasts, seem to me to have utterly ignored the mechanical and chemical conditions, which may produce want of coaptation of a corneal flap, and consequently imperfect healing. That suppuration may result from the forcible opening of the wound, when every precaution has been taken as to cleanliness, cannot be denied ; or that a constant agitation of the flaps, such as occurred in the case where the patient, an old lady, shook her head continuously for twenty-four hours, beginning immediately after the operation was performed, may do the same thing.

I have pursued various methods as to the after-treatment, but I have finally come down to leave the eye closed under the bandages for at least four or five days, whenever possible. I have never forsaken Graefe's bandage, except a very short period, when I used a black patch. I believe that better adapted than anything else for keeping the eye quiet, and that it is generally comfortable to the patient, and a great preventive of injury to the eyeball.

As the years have gone on, I have changed very much my ideas of the necessity of keeping the room free from light or of preventing the patient from moving or sitting up in bed. I allow much more latitude in that direction. I am inclined, however, to operate upon restless patients on the bed where the patient is afterward to lie.

I know of a very competent operator in Louisville, Kentucky, who allows some of his patients to go home twenty miles or more in a country wagon, and although I have had at least three successful cases that have undergone at least this much, I do not advise anything but a quiet recumbent position during the first few hours after an extraction of cataract by the flap method.

After inventing a method for the extraction of the lens in its capsule, which I practised almost exclusively for a year or so, I gave it up because it seemed to me, that its great supposed advantage did not exist, and because it is an operation much more difficult than what I now usually perform—simple extraction.[1]

Cocaine has but once failed me as an efficient local anæsthetic, and that was in an operation performed on March 2d of this year, and not included in this paper. This was in the case of an unruly and stupid old woman, for whom I performed a preliminary iridectomy with great difficulty, although her eye was fully anæsthetized. The house surgeon of the hospital, Dr. Black, for two weeks drilled her in the technique of bearing the operation, but all without avail. Ether was then given, and although with great care, she became asphyxiated and the radial pulse stopped, so that I was obliged to give up the ether, to give a dose of whiskey, use cocaine, and operate an hour later. The section was made with a moderate degree of difficulty, and just as I was beginning to congratulate myself she pushed out the lens and a small quantity of vitreous with her orbicularis. She has done well and has good vision, although she had delirium. Before presenting the general table I wish to add, that the statistics of but 204 are tabulated, because the 205th and 206th cases were not operated upon with any hope of restoring sight: one was for cosmetic reasons; the other, that of a poor young woman from the Blind Asylum with an atrophy of the optic nerve, certainly in one eye and probably in the other, at her urgent request.

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[1] In this operation I dislocated the lens with the back of the knife before finishing the section.

TABLE OF THE RESULTS OF THE EXTRACTION OF CATARACT IN 206 EYES.[1]

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
1	M. 55.	L.	Ether. Flap. Beer's knife. Iris wounded. 39 days.	Iridectomy.	$V=\frac{20}{200}$. Later, $V=\frac{20}{100}$.
2	M. 55. Same as No. 1.	R.	Ether. Flap. Beer's knife. 15 days.	Keratonyxis.	$V=\frac{20}{100}$.
3	M. 45.	L.	Lower section. Beer's knife. 20 days.	None.	$V=\frac{20}{100}$.
4	F. 60.	L.	Flap. Beer's knife. 18 days.	None.	$V=\frac{20}{70}$.
5	F. 70.	R.	Flap. Beer's knife. 28 days.	None.	$V=\frac{20}{200}$.
6	F. 50.	L.	Ether. After preliminary iridectomy. 25 days.	Iridectomy.	$V=\frac{12}{200}$.
7	M. 56.	R.	Lower section. Beer's knife. Iridectomy. 50 days.	None.	$V=\frac{20}{200}$.
8	M. 65.	L.	Ether. Lower flap. Beer's knife. 28 days.	None.	$V=\frac{20}{200}$.
9	M. 56. Same as No. 7.	L.	Ether. Lower section. Beer's knife. 21 days.	None.	$V=\frac{20}{70}$.
10	M. 30.	L.	Ether. Linear. 40 days.	Keratonyxis 3 times.	$V=\frac{20}{70}$.
11	F. 60.	L.	Flap with iridectomy. Beer's knife. 26 days.	Keratonyxis.	$V=\frac{20}{100}$.
12	F. 66.	R.	Liebreich. 45 days.	Keratonyxis.	$V=\frac{20}{100}$.
[2] 13	F. 60. Same as No. 11.	R.	Liebreich. Lens dislocated into vitreous. Wound reopened one week later and lens removed. 49 days.	Keratonyxis.	$V=\frac{20}{200}$. Later, $V=\frac{20}{70}$.
14	F. 67.	L.	Ether. Simple. 25 days.	Keratonyxis.	$V=\frac{20}{50}$.
15	M. 57. Paralysis agitans.	L.[2]	Liebreich. Iris wounded. Loss of vitreous. 90 days.	Keratonyxis 3 times.	$V=\frac{13}{200}$.
16	F. 57.	R.	Ether. Graefe. Iris wounded. 89 days.	Iridotomy.	$V=\frac{10}{200}$.
17	F. 53.	R.	Graefe. 23 days.	None.	$V=\frac{20}{70}$.
18	M. 67.	R.	Ether. Liebreich.		$V=\frac{20}{100}$.
19	F. 20.	R.	Ether. Graefe. Loss of vitreous. Globe collapsed.	None.	V=Counts fingers.
20	F. 75.	L.	Graefe. Loss of vitreous. 22 days.	None.	$V=\frac{14}{200}$.
21	F. 49.	L.	Ether. Section downward. Iridectomy. 20 days.	Keratonyxis.	$V=\frac{20}{100}$.
22	M. 75.	R.	Ether. Section downward. Iridectomy. Iris wounded. 20 days.	None.	$V=\frac{20}{100}$.
23	M. 59.	L.	Ether. Graefe.	None.	V=0. Kerato-irido-choroiditis.
24	F. 55.	R.	Graefe. Loss of vitreous. 33 days.	None.	V=Perception of light.
25	M. 75.	R.	Ether. Graefe. 35 days.	Declined.	V=Perception of light.

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[1] For much valuable and indispensable assistance in preparing these tables, I am glad to thank my chief of staff, Dr. Frank N. Lewis, Assistant-Surgeon in the Manhattan Eye and Ear Hospital.

[2] Transactions Am. Ophth. Society, 1873, page 69.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
26	M. 75. Same as No. 25.	L.	Ether. Graefe. 16 days.	None.	V=Perception of light.
27	F. 72.	L.	Ether. Graefe. 40 days.	None.	$V=\frac{20}{40}$.
28	M. 44.	R.	Ether. Graefe. 13 days.	None.	V=Counts fingers.
29	F. 50.	L.	Ether. Graefe. Iris wounded. 22 days.	None.	$V=\frac{20}{70}$.
30	M. 61.	R.	Lens in capsule. Loss of vitreous. 22 days.	None.	$V=\frac{20}{200}$.
31	F. 73.	L.	Ether. Graefe. Loss of vitreous. 31 days.	None.	V=Counts fingers.
32	M. 28.	R.	Ether. Graefe. 30 days.	Keratonyxis and iridotomy.	$V=\frac{20}{100}$.
33	F. 52.	R.	Ether. Graefe. 30 days.	None.	$V=\frac{20}{70}$.
34	M. 28. Same as No. 32.	L.	Ether. Lower section. Iridectomy. 15 days.	None.	$V=\frac{20}{100}$.
35	F. 28.	R.	Ether. Graefe. Iris wounded. 22 days.	None.	$V=\frac{20}{100}$.
36	M. 45.	L.	Ether. Lower section. Iris wounded. Iridectomy. 29 days.	None.	$V=\frac{20}{30}$.
37	F. 50.	L.	Ether. Lower section. Iris wounded. Iridectomy. 13 days.	None.	$V=\frac{20}{200}$.
38	M. 44.	R.	Ether. Graefe. Iris wounded. Loss of vitreous. 8 days.	None.	V=Perception of light.
39	F. 74.	L.	Ether. Graefe. Loss of fluid vitreous. 41 days.	Iridectomy.	V=Large objects.
40	F. 70.	R. Myopic.	Ether. Graefe. 40 days.	None.	$V=\frac{20}{100}$.
41	F. 70.	L.	Ether. Lower section. Iridectomy. 24 days.	None.	$V=\frac{20}{100}$.
42	F. 63.	R.	Ether. Graefe. 25 days.	Keratonxyis.	$V=\frac{20}{200}$.
43	M. 76.	R.	Ether. Lens in capsule. Loss of vitreous. 32 days.	None.	$V=\frac{20}{70}$.
44	M. 69. Good.	L. Senile. 1½ yrs.	Ether. Graefe. Loss of vitreous. Very unruly; sent home. 1 day.	None.	$V=\frac{20}{100}$.
45	F. 60. Good.	R. Inflammatory. V. F. imperfect.	Preliminary iridectomy 6 months before. 60 days.	None.	V=Perception of light.
46	M. 70. Good.	R. Senile.	Ether. Lens in capsule. 18 days.	None.	$V=\frac{20}{70}$.
47	M. 66. Good.	R. Senile.	Lens in capsule. Small iridectomy. 18 days.	None.	$V=\frac{20}{40}$.
48	M. 52. Alcoholic.	R. Senile. 2 yrs.	Ether. Lens in capsule. Iris wounded. Small iridectomy. Loss of vitreous. 14 days.	None.	$V=\frac{18}{200}$.
49	F. 58. Good.	L. Senile. 6 yrs.	Ether. Section downward. No iridectomy. 12 days.	None.	$V=\frac{8}{200}$.
50	F. 44. Good.	R. Senile. 9 yrs.	Ether. Lens in capsule. Loss of vitreous. 41 days.	None.	$V=\frac{20}{70}$. 6 mos. later, $V=\frac{20}{100}$.
51	F. 53. Good.	L. Senile. Corneal opacities.	Ether. Preliminary iridectomy. 15 days.		$V=\frac{15}{200}$.
52	M. 60. Poor.	R. Glaucomatous. 11 days. V. F. bad.	Ether. Iridectomy for glaucoma 11 days before.	None.	V=Perception of light.
53	F. 65. Good.	L. Senile.	Ether. Section downward. 36 days.	Keratonyxis.	$V=\frac{20}{100}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
54	F. 53. Good.	R. Senile. 8 yrs.	Ether. Preliminary iridectomy 6 months before. Lens in capsule. Loss of vitreous. 144 days.	None.	V=$\frac{1}{200}$. Irido-choroiditis.
55	M. 56. Good.	L. Senile. 1 yr.	Ether. Preliminary iridectomy. 27 days.	None.	V=$\frac{20}{100}$. 1 year later, V=$\frac{16}{200}$.
56	F. 77. Good.	R. Senile. 5 yrs.	Ether. Lens in capsule. 42 days.	None.	V=$\frac{8}{200}$.
57	F. 65. Good.	L. Senile. 4 yrs.	Cocaine. Preliminary iridectomy. Section downward. 30 days.	None.	V=$\frac{20}{200}$.
58	M. 61. Good.	L. Senile.	Cocaine. Lens in capsule. Section downward. 16 days.	None.	V=$\frac{20}{40}$.
59	F. 61. Good.	L. Senile. 3 yrs.	Cocaine. Preliminary iridectomy. Lens in capsule. Loss of vitreous. 22 days.	None.	V=$\frac{20}{100}$.
60	M. 72. Good.	R. Senile. 5 yrs. Perception poor.	Cocaine. Lens in capsule. Loss of vitreous. 23 days.	None.	V=No perception.
61	F. 50. Good.	L. Senile.	Cocaine. Lens in capsule. Loss of vitreous. 27 days.	None.	V=$\frac{20}{30}$.
62	F. 58. Good.	L. Inflammatory. 2 yrs. Posterior-synechia.	Cocaine. Preliminary iridectomy. 68 days.	Iridotomy.	V=$\frac{8}{200}$.
63	F. 58. Good.	R. Inflammatory. 2 yrs. Post.-synech.	Cocaine. Preliminary iridectomy. Part of lens removed. Loss of vitreous.		Enucleation.
64	F. 70. Good.	L. Senile. 5 mos.	Cocaine. Lens in capsule. Loss of vitreous. 57 days.	None.	V=$\frac{20}{200}$.
65	F. 50. Good.	R. Senile. 3 mos.	Cocaine. Lens in capsule. Eversion of flap. Loss of vitreous. 37 days.	None.	V=$\frac{20}{200}$.
66	F. 64. Good.	L. Senile. 3 mos.	Cocaine. Preliminary iridectomy. Lens removed by spoon. 34 days.	Iridectomy.	V=Perception of light.
67	F. 63. Good.	L. Senile. 2 yrs.	Cocaine. Lens in capsule. Iridectomy. Loss of vitreous. 52 days. 2 months later, hypopyon-iritis; pus absorbed by use of hot water. V remained $\frac{20}{70}$.	None.	V=$\frac{20}{70}$. Feb., 1890, V=$\frac{20}{70}$.
68	M. 64. Good.	L. Senile. 2 yrs.	Cocaine. Preliminary iridectomy. Lens in capsule. 33 days.	Keratonyxis.	V=$\frac{20}{70}$.
69	M. 58. Good. Same as No. 55.	R. Senile. 3 yrs.	Cocaine and ether. Liebreich. 28 days.	Keratonyxis.	V=$\frac{20}{200}$.
70	F. 54. Good.	L. Senile. Chronic conjunctivitis.	Ether. Lens in capsule. Section downward. Loss of vitreous. 24 days.		V=$\frac{20}{100}$.
71	M. 62. Good.	R. Senile. 2 yrs.	Cocaine. Lens in capsule. Loss of vitreous. 16 days.	None.	V=$\frac{20}{200}$.
72	M. 55. Good.	R. Senile. 1 yr.	Cocaine. Preliminary iridectomy 5 days before. 64 days. Hypopyon-iritis.	Paracentesis.	V=Perception of light.
73	M. 53. Good.	L. Senile. 6 yrs. V.F. imperfect	Cocaine. Lens in capsule. Loss of vitreous. 54 days.	None.	V=$\frac{20}{200}$.
74	M. 55. Same as No. 72.	L. Senile. 1 yr.	Cocaine. Lens in capsule. Iris wounded. 29 days.	None.	V=$\frac{20}{40}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
75	F. 64. Good.	L. Senile. 5 yrs.	Cocaine. Lens in capsule. Iris wounded. Loss of vitreous. Later flap found dislocated. 74 days.	None.	V=Counts fingers.
76	M. 55. Good.	L. Senile.	Cocaine. Preliminary iridectomy. Loss of vitreous before section completed. Lens dislocated into vitreous. 27 days.	None.	$V=\frac{2}{200}$.
77	M. 59. Good.	L. Senile. 6 mos.	Cocaine. Section downward. Capsule ruptured by knife. Iris wounded. 22 days.	Keratonyxis.	$V=\frac{20}{70}$. 4 mos. later, keratonyxis. $V=\frac{20}{100}$.
78	F. 75. Good.	R. Senile.	Cocaine. Lens in capsule. Iridectomy. Loss of vitreous. Eversion of flap. 44 days.	Keratonyxis.	$V=\frac{20}{200}$ 2 mos. later, $V=\frac{20}{200}$.
79	F. 62. Good.	L. Senile. Chr. glaucoma.	Ether. Preliminary iridectomy. 57 days.	Keratonyxis.	$V=\frac{20}{200}$. 4 yrs. later, $V=\frac{20}{200}$.
80	F. 52. Good.	L. Senile. Retino-choroiditis.	Ether. Preliminary iridectomy 9 days before. Lens in capsule. 22 days.	None.	$V=\frac{20}{100}$.
81	M. 74. Good.	R. Senile. 6 yrs.	Cocaine. Lens in capsule. 15 days.	None.	$V=\frac{20}{30}$. 2 years later, $V=\frac{20}{30}$.
82	F. 60. Good.	L. Senile. 2 yrs.	Cocaine. Lens in capsule. Iridectomy by knife. 23 days.	None.	$V=\frac{20}{50}$.
83	M. 59.	L. Senile.	Cocaine. Lens in capsule. Iris wounded. Cornea collapsed. 35 days.	None.	$V=\frac{20}{200}$. 4 years later, V=Perception of light.
84	F. 67. Good.	R. Senile. 3 yrs.	Cocaine. Lens in capsule. Section downward. 22 days.	None.	$V=\frac{20}{200}$. 1 year later, $V=\frac{20}{30}$.
85	F. 63. Good.	L. Senile. 6 yrs.	Cocaine. Simple. Iris wounded. 44 days.	Iridectomy.	$V=\frac{20}{30}$.
86	M. Good.	R. Senile. Choroiditis.	Cocaine. Lens in capsule. Iris wounded. 17 days.	None.	$V=\frac{20}{100}$.
87	M. 65. Fair.	L. Senile.	Cocaine. Lens in capsule. Iris wounded. Vitreous lost. Died 38 days later.	None.	$V=\frac{20}{100}$, estimated.
88	M. 58. Good.	L. Senile.	Cocaine. Simple. 37 days.	Keratonyxis.	$V=\frac{20}{40}$.
89	F. 53. Good.	L. Senile. 5 yrs. Chr. conj.	Cocaine. Förster 1 year before. 32 days.	Keratonyxis.	$V=\frac{20}{40}$.
90	M. 69. Good.	R. Senile. 2 yrs.	Cocaine. Simple. Piece of iris excised. 29 days.	Keratonyxis.	$V=\frac{20}{50}$.
91	M. 64. Good.	L. Senile. 3 yrs.	Cocaine. Simple. 42 days.	Keratonyxis.	$V=\frac{20}{200}$. 2 yrs. later, $V=\frac{20}{100}$.
92	F. 56. Good.	L. Senile. 3 mos.	Cocaine. Simple. 23 days.	None.	$V=\frac{20}{40}$.
93	F. 60. Fair.	R. Senile. Chr. conj.	Cocaine. Preliminary iridectomy. 42 days.	Keratonyxis.	$V=\frac{20}{50}$. 2½ yrs. later, $V=\frac{20}{30}$.
94	M. 70. Good.	L. Senile. 6 mos.	Cocaine. Simple. 31 days.	None.	$V=\frac{20}{200}$. 1 yr. later, $V=\frac{20}{50}$.
95	F. 79. Fair.	L. Senile. 5 yrs.	Cocaine. Simple. 43 days.	None.	Panophthalmitis, following suppuration of flap.
96	F. 46. Good.	L. Senile. 5 yrs.	Cocaine. Simple. 22 days.	None.	$V=\frac{20}{20}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
97	F. 70. Fair.	R. Senile. 2 yrs.	Cocaine. Lens in capsule. 15 days.	None.	$V=\frac{20}{40}$.
98	F. 70. Fair.	R. Senile. 3 yrs.	Cocaine. Simple. Patient unruly. 17 days.	None.	$V=\frac{20}{100}$.
99	F. 74. Fair.	R. Senile. 3 yrs.	Cocaine. Lens in capsule. Vitreous lost. 26 days.	None.	Suppuration of cornea senile. Dementia. Died.
100	M. 63. Alcoholic.	R. Senile. 6 mos.	Cocaine. Lens in capsule. Vitreous lost. 32 days.	None.	$V=\frac{20}{50}$. 2 years later, $V=\frac{20}{40}$.
101	M. 75. Good.	L. Senile.	Cocaine. Simple. Downward. 77 days.	Iridotomy.	V=Perception of light. Suppuration corneal edge.
102	F. 67. Poor.	L. Senile.	Cocaine. Graefe. Vitreous lost. Collapse of cornea. 46 days.	Keratonyxis.	$V=\frac{20}{100}$, later $V=\frac{20}{200}$.
103	M. 63. Good.	L. Senile.	Cocaine. Simple. 63 days.	Keratonyxis.	$V=\frac{7}{200}$. Suppuration of wound.
104	M. 30. Brother of No. 32.	R. Congenital.	Cocaine. Preliminary iridectomy. 21 days.	Keratonyxis.	$V=\frac{20}{100}$.
105	M. 68. Good.	L. Senile. 2 yrs.	Cocaine. Simple. 46 days.	None.	$V=\frac{20}{100}$.
106	M. 67. Good.	R. Senile. 1½ yrs.	Cocaine. Simple. 14 days.	None.	$V=\frac{20}{50}$.
107	F. 50. Good.	L. Senile. 7 mos.	Cocaine. Simple. 19 days.	None.	$V=\frac{20}{200}$.
108	M. 62. Good.	R. Senile. 8 mos.	Cocaine. Simple. 31 days.	Keratonyxis.	$V=\frac{2}{200}$. Kerato-iritis.
109	F. 60. Good.	R. Senile.	Cocaine. Simple. 38 days.	Iridotomy.	V=Counts fingers. 6 mos. later, $V=\frac{12}{200}$.
110	M. 80. Good.	R. Senile. 2 yrs.	Cocaine. Simple. 31 days.	Iridotomy.	V=Counts fingers. Kerato-iritis.
111	F. 50. Good.	L. Senile. 4 yrs. V.F. imperfect	Cocaine. Graefe. 29 days.	None.	$V=\frac{7}{200}$.
112	M. 48. Fair.	L. Senile. 4 yrs. Projection slow.	Cocaine. Graefe. 36 days.	Keratonyxis.	V=Perception of light.
113	M. 65. Good.	L. Senile. 6 mos.	Cocaine. Graefe. 27 days.	None.	Phthisis bulbi. Enucleation.
114	F. 64. Fair.	R. Senile. 2½ yrs.	Cocaine. Graefe. Unruly; removed bandage herself. 28 days.	None.	$V=\frac{20}{40}$.
115	F. 70. Good.	R. Senile. 5 yrs.	Cocaine. Preliminary iridectomy. 21 days.	Keratonyxis.	V=Perception of light. Hypopyon - iritis.
116	F. 53. Good.	R. Senile. 3 yrs.	Cocaine. Graefe. Vitreous lost. 41 days.	None.	Phthisis bulbi.
117	F. 70. Good.	L. Senile. 3½ yrs.	Cocaine. Graefe. 21 days.	None.	$V=\frac{20}{100}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
118	F. 50. Good.	R. Senile. 2 yrs. Eye always amblyopic.	Cocaine. Graefe. Vitreous lost. 39 days.	Keratonyxis.	$V=\frac{2}{200}$. Detachment of retina 9 mos. later.
119	F. 28. Good.	R. Congenital Small cornea	Cocaine. Preliminary iridectomy. 9 days.	Keratonyxis.	$V=\frac{5}{200}$. Later, $V=\frac{12}{200}$.
120	F. 66. Fair.	R. Senile. 2 yrs.	Cocaine. Graefe. 14 days.	None.	$V=\frac{20}{50}$.
121	M. 48. Good.	L. Senile. Dacrocystitis.	Cocaine. Preliminary iridectomy. 21 days.	None.	$V=\frac{20}{70}$.
122	M. 49. Poor.	L. Senile. Hypermature.	Cocaine. Graefe. 46 days.	None.	$V=\frac{15}{200}$.
123	F. 28. Same as No. 119.	L. Congenital Small cornea	Ether. Linear. 10 days.	None.	$V=\frac{20}{200}$.
124	M. 65. Same as No. 113.	R. Senile. Inflammatory. V F. poor.	Ether. Preliminary iridectomy. 18 days.	None.	V=Perception of light.
125	M. 62. Same as No. 83.	R. Senile. 3 yrs.	Cocaine. Preliminary iridectomy. 45 days.	None.	$V=\frac{2}{200}$.
126	M. 13. Good.	R. Traumatic. 6 mos.	Cocaine. Linear. 19 days.	Iridotomy.	$V=\frac{20}{100}$.
127	F. 50. Same as No. 118.	L. Senile. Myopic. Not mature.	Cocaine. Simple. Vitreous lost. 44 days.	Iridotomy.	$V=\frac{9}{200}$ without a glass.
128	M. 73. Good.	R. Senile. 8 yrs.	Cocaine. Lens in capsule unintentional. 26 days.	None.	$V=\frac{20}{200}$.
129	M. 69. Same as No. 68.	R. Senile. 8 yrs.	Cocaine. Simple. 14 days.	None.	$V=\frac{20}{40}$. Later, $V=\frac{20}{20}$.
130	M. 32. Same as No. 104.	L. Congenital	Cocaine. Preliminary iridectomy. 17 days.	None.	$V=\frac{7}{200}$. Later, $V=\frac{20}{30}$.
131	F.	L. Capsular.	Cocaine. Simple. 24 days.	None.	$V=\frac{20}{200}$.
132	F. 60. Fair.	L. Senile.	Ether. Lens in capsule. 18 days.	Iridectomy.	$V=\frac{20}{100}$.
133	F. 22.	R. Congenital. Nerve of other eye atrophic. Feeble perception.	Ether. Graefe. 17 days.	None.	V=Perception of light. Nerve atrophied
134	M. 13. Good.	R. Traumatic.	Cocaine. Linear. 15 days.	Keratonyxis.	$V=\frac{20}{200}$.
135	M. 20.	R. Congenital.	Cocaine. Linear.	None.	$V=\frac{5}{200}$. Later, $V=\frac{10}{200}$.
136	M. 20. Same as No. 135.	L. Congenital.	Cocaine. Linear. 48 days.		$V=\frac{10}{200}$.
137	M. 49. Poor.	L. Senile.	Cocaine. Graefe. 57 days.	None.	$V=\frac{20}{100}$.
138	F. 22.	R. Soft. 10 yrs.	Ether. Linear. 18 days.	None.	$V=\frac{20}{100}$.
139	M. 37. Good.	R. Traumatic. 12 days. Lens under conjunctiva.	Cocaine. Opening of conjunctiva. Lens removed with spoon. 22 days.	None.	$V=\frac{18}{200}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
140	M. 45. Good.	L. Dislocated lens forward.	Cocaine. Linear. 17 days.	None.	$V=\frac{20}{200}$.
141	M. 47.	R. Traumatic.	Cocaine. Linear. 43 days.	Keratonyxis. 3 times.	$V=\frac{20}{50}$.
142	M. 51. Good.	L. Senile.	Flap, lower section. Beer's knife.	None.	$V=\frac{20}{70}$.
143	M. 51. Same as No. 142.	R. Senile.	Flap, lower section. Beer's knife. Lens dislocated into vitreous.	None.	$V=\frac{20}{200}$.
144	M. 60. Asthma.	R. Senile. 2 yrs.	Flap. Beer's knife. 20 days.	None.	$V=\frac{20}{50}$. 20 yrs. later, V same.
145	M. 65. Fair.	L. Senile.	Lens dislocated and removed with hook. 28 days.	None.	$V=\frac{16}{200}$.
146	M. 59. Good.	R. Senile.	Flap. Beer's knife. Iridectomy. 14 days.	None.	$V=\frac{20}{40}$, observed for 10 yrs. after.
147	F. 76. Good.	L. Senile. 7 yrs.	Lower section. Beer's knife. Iridectomy. 21 days.	None.	$V=\frac{20}{100}$.
148	F. 76. Same as No. 147.	L. Senile. 7 yrs.	Upper section. Beer's knife. Iridectomy. 21 days.	None.	$V=\frac{20}{200}$. Opacity cornea.
149	M. 60. Good.	R. Senile.	Lower section. Beer's knife. 21 days.	None.	$V=\frac{10}{200}$.
150	M. 60. Same as No. 149.	R. Senile.	Upper section. Beer's knife. 21 days.	None.	$V=\frac{20}{200}$, observed 20 years.
151	M. 63. Same as No. 144.	L. Senile.	Flap. Beer's knife. 18 days.	None.	$V=\frac{10}{70}$. Later, $V=\frac{20}{40}$.
152	M. 52. Good.	R. Senile.	Ether. Simple. Beer's knife. 28 days.	None.	V=objects.
153	F. 65. Good.	R. Senile.	Graefe. Patient very restless after, and kept head moving from side to side. Suppuration of cornea. 4 weeks.	None.	V=Quantitative Perception.
154	M. 65. Good.	L. Senile.	Liebreich.	Keratonyxis.	$V=\frac{20}{100}$. 10 yrs. later, $V=\frac{20}{70}$.
155	M. 76. Good.	R. Senile. 2 yrs.	Ether. Graefe.	Keratonyxis.	$V=\frac{20}{50}$. Later, $V=\frac{20}{70}$.
156	F. 72. Good.	Senile.	Graefe.		$V=\frac{20}{70}$.
157	M. 87. Good.	L. Senile. 12 yrs.	Lower section. Lens removed with spoon. Vitreous lost.	Keratonyxis.	$V=\frac{18}{200}$.
158	M. 72. Good.	L. Senile. 7 yrs.	Ether. Graefe.	None.	$V=\frac{20}{70}$, observed 10 yrs. later
159	F. 67. Insane.	R. Senile. 4 yrs.	Ether. Lens in capsule. 4 weeks.	None.	$V=\frac{20}{100}$.
160	M. 68. Good.	R. Senile. 2 yrs. Myopic.	Small simple corneal section. Vitreous lost.	Iridectomy. Iridotomy. Keratonyxis.	$V=\frac{9}{200}$. Later, $V=\frac{10}{200}$.
161	F. 74.	R. Senile.	Lens in capsule,		$V=\frac{20}{200}$.
162	F. 61. Good.	L. Senile.	Ether. Section downward. Iridectomy.	None.	$V=\frac{20}{70}$, observed 10 yrs. after
163	M. 28. Brother of No. 130.	R. Congenital. Slowly developing.	Ether. Lens in capsule. Two needlings previous without effect. 2 months.	Iridotomy.	$V=\frac{20}{70}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
164	M. 28. Same as No. 163.	L. Congenital.	Ether. Graefe. Small corneal section. Iridectomy. 3 months.	Iridotomy.	$V=\frac{10}{200}$.
165	M. 72. Good.	R. Senile.	Ether. Lens in capsule. Iris cut. Vitreous lost from coughing. Imperfect etherization. 3 months.	None.	Panophthalmitis.
166	F. 62. Feeble.	L. Senile.	Section downward. Iridectomy.	None.	$V=\frac{20}{70}$. 3 mos. later, irido-choroiditis & $V=\frac{18}{200}$.
167	F. 66. Chr. bronchitis.	R. Senile. 2 yrs.	Graefe. Upper section, Iridectomy.	Keratonyxis twice.	$V=\frac{20}{200}$.
168	F. 70. Same as No. 167.	L. Senile. 7 yrs.	Cocaine. Förster 1 year before. Lens in capsule.	None.	$V=\frac{20}{40}$, observed 4 yrs. later.
169	M. 75. Good.	L. Senile. 2 yrs. Lach. catarrh.	Ether. Lower section. Iridectomy. Vitreous lost. 35 days.	None.	V=Perception of light.
170	M. 60. Alcoholic.	R. Senile. 1 yr. Myopic.	Ether. Graefe. 21 days.	None.	$V=\frac{20}{70}$. Detachment of retina 4 years later.
171	M. 51. Son of No. 155.	L. Senile. 3 yrs. Myopic.	Ether. Lens in capsule. Vitreous lost. 21 days		$V=\frac{20}{50}$, observed 7 yrs. after.
172	M. 36. 2d son of No. 155. Delicate.	R. Senile. 4 yrs.	Cocaine. Lens in capsule. 21 days.	Keratonyxis.	$V=\frac{20}{50}$.
173	F. 59. Good.	Senile.	Simple. Beer's knife.	Keratonyxis.	$V=\frac{15}{200}$.
174	F. 53. Good.	R. Senile. 4 yrs.	Cocaine. Lens in capsule. Section downward. 21 days.	None.	$V=\frac{20}{100}$. Later, $V=\frac{20}{70}$
175	F. 60. Poor.	R. Senile. Glaucomatous.	Ether. Graefe.	None.	$V=\frac{20}{70}$. 4 years later, $V=\frac{3}{200}$.
176	M. 65. Bronchitis. Gout.	R. Senile.	Cocaine. Lens in capsule. Hit his eye after operation. Suppuration. 6 wks.	None.	Phthisis bulbi.
177	F. 44. Daughter of No. 144. Delicate.	R. Senile. 5 yrs.	Cocaine. Simple. 28 days.	Keratonyxis twice.	$V=\frac{20}{40}$.
178	F. 59. Feeble.	R. Senile. 6 mos.	Cocaine. Simple. Vitreous lost. 28 days.	None.	$V=\frac{20}{70}+$.
179	F. 93. Good.	R. Senile.	Cocaine. Lens in capsule. 28 days.	None.	$V=\frac{20}{70}$, observed 5 yrs. later.
180	M. 82. Good.	R. Senile. 2 yrs.	Cocaine. Preliminary iridectomy. Cornea collapsed.	None.	$V=\frac{20}{70}$. Later, $V=\frac{8}{200}$.
181	F. 60. Good.	L. Senile. 6 mos.	Cocaine. Simple. Capsule divided with knife. 28 days.	None.	$V=\frac{20}{50}$.
182	F. 75. Good.	L. Senile. 3 yrs.	Cocaine. Preliminary iridectomy. Lens in capsule.	Iridotomy.	$V=\frac{20}{200}+$.
183	M. 39. Good.	L. Senile. 1 yr.	Cocaine. Linear. 21 days.	None.	$V=\frac{20}{30}$. Later, $V=\frac{20}{30}$.

No.	Sex. Age. Health.	Eye. Quality. Duration.	Anæsthetic. Operation. Length of Treatment.	Secondary Operation.	Result.
184	F. 67. Fair.	R. Senile. 11 yrs.	Cocaine. Lens in capsule. 2 months.	Iridectomy of prolapsed iris.	Enucleation. Panophthalmitis.
185	M. 76. Good.	R. Senile. 2 yrs.	Cocaine. Preliminary iridectomy. Lens in capsule. 21 days.	None.	V=$\frac{20}{70}$, observed 3 yrs. later.
186	F. 64. Feeble. Same as No. 178.	L. Senile. 4 yrs.	Cocaine. Simple. Suppurative iritis after wound had closed and V was good. 3 months.	None.	V=objects.
187	M. 38. Good.	R. Soft. 5 yrs.	Cocaine. Linear with iridectomy. 2 months.	Keratonyxis.	V=$\frac{20}{70}$.
188	F. 82. Good.	L. Senile. 10 yrs.	Cocaine. Simple. Much soft cortical left.	Iridotomy and linear section.	V=$\frac{10}{200}$.
189	M. 27. Good.	L. Soft. 1 yr.	Cocaine. Linear after capsulotomy. 21 days.	Keratonyxis twice. Observed for 3 years.	V=$\frac{20}{20}$+.
190	M. 27. Same as No. 189.	R. Soft. 1 yr.	Cocaine. Linear after capsulotomy. 21 days.	Keratonyxis.	V=$\frac{20}{70}$. Later, V=$\frac{20}{30}$.
191	F. 60. Good.	L. Senile. 1½ yrs.	Ether. Graefe.	None.	V=$\frac{9}{100}$.
192	F. 58. Poor.	Senile. 3 yrs.	Ether. Graefe.	None.	V=$\frac{20}{200}$.
193	F. 55. Good.	R. Senile. 4 yrs.	Lens in capsule. 9 days.	None.	V=Counts fingers.
194	M. 82. Good.	R. Senile. 2 yrs.	Graefe. 5 days.	None.	V=Counts fingers.
195	F. 81. Good.	R. Senile. 12 yrs.	Lens in capsule.	None.	V=$\frac{20}{100}$.
196	F. 41. Good.	R. Senile. 4 yrs.	Graefe. Vitreous lost.	None.	V=$\frac{7}{200}$.
197	F. 63. Good.	R. Senile. 6 yrs. V.F. imperfect	Graefe. 6 days.	None.	V=$\frac{2}{200}$.
198	M. 69. Good.	L. Senile.	Section downward.	None.	V=$\frac{20}{30}$.
199	F. 70. Good.	L. Senile. 1 yr.	Section downward.	None.	V=$\frac{20}{70}$.
200	M. 60. Poor.	L. Senile. Tremulous iris.	Section downward. Vitreous lost. Lens removed with hook.	None.	V=$\frac{20}{100}$.
201	M. 73. Good.	L. Senile. 5 yrs.	Section downward.	None.	V=$\frac{20}{70}$.
202	M. 54. Poor.	L. Senile. 2 yrs.	Lens in capsule.	None.	V=Perception of light.
203	M. 72. Good.	R. Senile. 15 yrs.	Ether. Lens in capsule.	None.	V=$\frac{20}{20}$.
204	M. 76. Good.	L. Senile. 6 yrs.	Ether. Graefe. 16 days.	None.	V=$\frac{20}{40}$.
205	F. 61. Poor. Same as No. 109.	L. Senile. 3 yrs.	Cocaine. Graefe.	None.	V=$\frac{20}{30}$.
206	M. 44. Good.	R. Soft. Inflammatory. Myopic. Feeble perception.	Cocaine. Linear and suction with Panas syringe.	None.	V=Perception of light.

CONDITION OF EYE BEFORE OPERATION.

Myopic Nos. 40, 127, 160, 170, 171.
V. F. imperfect " 73, 111, 112, 197.
Glaucomatous " 52, 175.
Choroiditis " 80, 86.
Inflammatory " 45, 62, 63, 124, 206.
Atrophy of optic nerve No. 133.
Amblyopic " 118.
Traumatic, 126, 134, 141.
Traumatic and dislocated, 139 and 140.
Congenital, 104, 119, 123, 130, 133, 135, 136, 163, and 164.
Capsular, 131.
Soft, 138 and 189.

TABLE SHOWING THE GENERAL RESULTS.

	No. of Cases.	
Vision $\frac{20}{20}$	5	
" $\frac{20}{30}$	10	
" $\frac{20}{40}$	16	
" $\frac{20}{50}$	10	
" $\frac{20}{70}$	28	
" $\frac{20}{100}$	34	
" $\frac{20}{200}$	33	
" $\frac{15}{200}$	7	
" $\frac{10}{200}$	17	
" $\frac{5}{200}$	7	
		167
" Counts fingers,	11	
" Perception of light,	17	
No vision,	11	39
		206

160 patients had one eye operated upon = 160 operations
23 " " both eyes " " 46 "

Total, 183 patients 206 operations

Cases No. 133, and No. 206 being excluded, of the remaining 204 cases, 167, or 82 %, were successful ; 37, or 18 %, were unsuccessful.

94 males. 89 females.

KIND OF OPERATION.	VISION.														
	$\frac{20}{20}$	$\frac{20}{30}$	$\frac{20}{40}$	$\frac{20}{50}$	$\frac{20}{70}$	$\frac{20}{100}$	$\frac{20}{200}$	$\frac{15}{200}$	$\frac{10}{200}$	$\frac{5}{200}$	Count Fingers,	Perception of Light,	No. Vision,	Total,	
Liebreich,					2	2	1	1						6	No failure.
Simple, Beer's knife,			1	1	2	2	2	2			1			11	1 failure.
Simple, Beer's knife lower section,					2		3							5	No failure.
Simple, Graefe's knife,	2	1	4	4		3	2		5	1	2		1	25	3 failures.
Simple, Graefe's knife lower section		1			1	3	2		2			1		10	1 failure.
With iridectomy, Beer's knife,			1			2	1							4	No failure.
With iridectomy, downward,		1			2	4	2					1		10	1 failure.
After preliminary iridectomy,		2	2		4	3	3	1	4	2	1	6	1	29	8 failures.
Graefe,		1	3	1	7	6	4	3	3	4	5	6	4	47	15 failures.
Lens in capsule,	1	3	5	3	6	7	9		1		2	2	5	44	9 failures.
Linear,	2	1		1	2	2	3		2			1		14	1 failures.
For lens dislocated under conjunctiva,							1							1	
Total,	4	11	16	10	28	34	33	7	17	7	11	17	11	206	

It may be justly said of these statistics, that they have been made up in the light of years of knowledge of the subsequent history of the cases, and, if the eye has been lost after a few months, or within any period to entitle it to be classed as within the proper time of the operation, the fact has been stated. Some of them had better vision within a month of the operation than they finally secured, and they would, in some instances, have been the better for a secondary operation, which they declined or did not wish for, being content with useful vision—that is to say, enough to go around with and to help themselves. The vast majority of the individuals were in the poorer walks of life, who are notoriously not exacting about their visual power, $\frac{20}{100}$ or $\frac{20}{70}$ being very satisfactory to them.

The cases of suppuration of the flap were, Nos. 23, 26, 95, 99, 101, 103, 113, 134, 157 of the table, all in sequence, except Nos. 134 and 157, which belong between Nos. 26 and 95 in order of time. Two, 95 and 99, were lost within a short time of each other at the hospital.

The eye was lost from suppuration of the cornea in 9 cases. From the patient's own negligence, in two certainly of the 37 cases of failure.

THE CASES WHICH DID NOT LEAVE THE PATIENTS WITH USEFUL VISION.

An analysis of some of these may be interesting, because I think it will be generally conceded that it is important to determine the causes of failure.

A middle-aged man in easy circumstances and of very full habit. He had a successful operation on the other eye. The technique of the operation was good, but iridochoroiditis set in, blocked up the pupil, and left him with simply perception of light. I never had an opportunity to perform any other operation, or it is probable that he would have been improved. The operation was performed in rather a poor hotel, but probably the comforts he had there were as many as he had at home.

A man, aged fifty-four. There was a good projection, but the cataract was traumatic, the eyeball having been injured by concussion. The other eye was lost from a wound by a knife; but there was no sympathetic inflammation. The prognosis was not good, and the eye ended where it began, with perception of light. I am unable to say in exactly what condition the choroid was.

A country woman, of about seventy years of age. This case has already been referred to. Here also the operation was without accident. It was performed with Beer's knife; no anæsthetic was used, and the patient went through the operation very well, having apparently nerved herself up to it. But she immediately began to move her head with a regular motion from side to side, as if she had a disease of the medulla. She displayed other symptoms of intense nervous excitement. Suppuration of the flap set in on the third day, and the eye was lost.

I conceive that the suppuration here was induced by the movements of the eye, communicated from the head, and that the inflammation was essentially traumatic in origin. The reason I have not been able to give my full assent to the Listerian doctrines of sepsis as the first cause in all cases of suppurating processes may be found in such cases as this one. I do not imagine that any ante-precautions in the face of this nervous excitement, producing a mechanical irritation, would have prevented suppuration. Coaptation of the flaps is essential, we all admit, to healing. This was prevented in this case by what has been detailed.

A man of seventy years. This case I had watched for years, with slowly developing cataract, he having had a serious attack of serous iritis, during the course of the time. His eye, however, had recovered perfectly, and was white, with good tension, good field ; when I operated upon him there were few synechiæ. He was an exceedingly anxious, fretful old man, who took ether badly, and just as the section was being finished he was attacked by coughing, the lens was extracted with difficulty, the vitreous began to come out and was fastened in the wound. The eye was lost from the first and after a tedious period of inflammation from irido-choroiditis. He died without an operation on the fellow-eye, some four years after.

A man, aged seventy-five, in good health, with a slight lachrymal catarrh. The wound closed, but was infected by inflammation of the sac, and the eye was lost, not by suppuration, but from plastic inflammation, probably by this infection. I subsequently operated upon the other eye, removing it in its capsule with success.

Another case of failure was that of a poorly nourished man of sixty-five years of age (case 176). The right lens was opaque ; there was a good visual field. In the left eye there was no absorption of light ; the lens was cataractous and dislocated. The right lens was removed in its capsule, cocaine being used. There was no difficulty met with in the extraction, no trouble of any kind. It was a beautiful operation, which was witnessed by his family physician, Dr. Vosburg, of this city. The eye was bandaged five days afterward, the patient not having suffered from pain or discomfiture. The cornea was found to be hazy ; hypopyon existed. The eye was lost by panophthalmitis. He was said to have struck his eye. The patient was gouty and had a cough. An attack of gout in the feet occurred during his first days of treatment. It seems to me that in this case the general nutrition possibly the injury, and not defects in the technique, were the provoking causes of the disease of the cornea.

Two cases of suppuration of the cornea leading to total loss were 95 and 99 of the table. Both occurred in very feeble old women; one died soon after she left the hospital, having senile dementia, the other had large ulcers on the leg. The hospital was just then, I thought, in not a good condition, for erysipelas had been in the wards.

SUBSEQUENT DETACHMENT OF THE RETINA.

One of the interesting but ultimately unfortunate cases, No. 170 of the table, occurred in an intemperate gentleman of sixty years, upon whom I performed Graefe's operation and got a good result. Vision, $\frac{20}{100}$, which afterwards, I improved to $\frac{20}{70}$. Some four years after my operation he suffered a detachment of the retina in that eye. This occurred in London, and Dr. Brudenell Carter operated upon the detachment by scleral puncture, without success; he also operated on the left eye for cataract. The vision of that eye had remained good when I saw him some months after the operation.

In another case, No. 118 of the series, detachment of the retina occurred nine months after the operation. The eye was myopic, and with marked choroiditis disseminata. The second eye, also an unsound one, was operated upon (No. 127 of the table) and fair vision resulted.

CHRONIC GLAUCOMA. GOOD RESULT.

Among the remarkable cases of the series is that of a lady of sixty years of age, in very reduced circumstances, who consulted me at the Manhattan Eye and Ear Hospital in 1881 for symptoms which were shown to be dependent upon chronic glaucoma of each eye. Her vision was then $\frac{15}{200}$ with the right eye, and $\frac{20}{20}$ with the left. She was treated by the use of eserine. In 1885 she appeared at my office with her vision reduced to quantitative perception in the right eye, and to $\frac{20}{60}$ in the left. Besides increased tension, she then had cataract in each eye. There was a general milky white opacity in each eye, but the reddish glare of the fundus could be seen through it; the tension was positively increased on the right side, less positively so on the left. On this point Dr. Emerson and I differed, he thinking it was positively increased, while I was not certain. After a preliminary iridectomy in December, 1886, the lens was removed under ether in October, 1887, with a subsequent needling through a tedious course, considerable cortex having been left behind, the wound not being quite large enough; $\frac{22}{100}$ vision, amply sufficient for her to go about everywhere alone, resulted.

I consider this a very fortunate case, for one eye was entirely lost by chronic glaucoma, and the other, although having probably the same affection with cataract supervening, has been saved for a period of more than five years with comfortable vision. Her vision has remained four years, as above stated.

UNSUCCESSFUL ATTEMPT TO REMOVE LENS IN CAPSULE.

One of the cases in which a certain degree of useful vision was obtained ($\frac{2}{70}$), but which was very unsatisfactory to the patient, and for which he blamed me very much, was that of a gentleman of eighty-one. A preliminary iridectomy was performed, because the cataract was very slow in ripening, I having watched it for years. The operation was performed under cocaine, and I attempted to remove the lens in its capsule, but it fell backward into the vitreous humor, and I could not secure it until I had lacerated the capsule, and extracted it in that way. There was a very slight loss of vitreous. The patient made a slow recovery, and capsulotomy was performed. There were floating bodies in his vitreous, and his vision was moderate, as stated above.

This patient was operated upon three years ago. He is still living.

This and the preceding case emphasize the opinion, which I believe to be correct, that slowly maturing cataracts are comparatively bad cases for operation. Such cases always have choroids that are not entirely healthy, or at least not nearly so healthy as those of cataracts that mature steadily and regularly. It is no doubt true, as a rule, that the nuclear cataract is the one offering the best prognosis, and that those where the cortex is chiefly affected are the slowly ripening ones; but it is a rule with exceptions.

Delirium has occurred in five cases. In one, that of a lady of ninety-three years of age we were obliged to give up all ideas of keeping the eyes even shaded (the operation—the extraction of the lens in its capsule—was witnessed by Dr. F. A. Burrall, her family physician, and the delirium, which lasted for some weeks, described by him in the *Medical Record*). We kept the room dark, or nearly dark, and allowed the old lady her own way under a nurse, and she made an excellent recovery. I visited her when she was ninety-six years old. She was able to sit up and receive her friends and read, so far as her enfeebled intellect would allow. She lived more than three years after the operation.

HOME-SICKNESS ; BAD SUBSEQUENT CARE ; GOOD RESULT.

The homesickness of one hospital patient was so great that I was sent for the evening after the operation, and I found the house surgeon, assistants, nurses, and matron all standing guard over a stalwart Irishman, who insisted on going home and seeing the old woman. He had already made havoc with the bandages. I sent him home and sent some one to visit him every day, and endeavored in every way to keep the eye protected. But in spite of us he was found shaving, looking into the glass with both eyes, a few days after the operation. His surroundings were of the humblest, and he had none of the advantages of the hospital, but he made a good recovery and had good vision—$\frac{20}{70}$.

GLAUCOMA.

In one case acute glaucoma supervened upon ordinary senile cataract, and I removed the lens to the immediate relief of the symptoms of glaucoma. The eye was very deeply sunken ; the operation was difficult and performed under cocaine. She had $\frac{20}{60}$ for some years. A membrane formed, but she declined an operation. Her vision was reduced by the membrane to about $\frac{10}{200}$.

In another, that of a lady of seventy-nine, glaucoma appeared in the fellow-eye while the patient was under treatment days after the lens had been extracted. She had chronic Bright's disease. The operation was complicated by senile entropion, but the lens was extracted in its capsule without difficulty, a preliminary iridectomy having been performed. The vision became $\frac{20}{100}$, with the ability to read fine type. When acute glaucoma supervened, I performed an iridectomy on the fellow-eye. I am not able to get any history of the patient later than a year after this, when she remained about the same.

Case 184 of the table was a promising one, that finally was totally lost. The patient, a female, was sixty-seven years of age. The lens was removed in its capsule, the wound healed in five days, and the patient had sharp vision for several days after.

Prolapse of the iris occurred at the 12th day. It was excised, but suppurative hyalitis set in, and the eyeball was enucleated. I have always believed that this patient interfered with her own eye by rubbing it, and that the prolapse was thus caused.

I operated in one case on a lady of sixty-seven, who was generally of sound mind, but who was subject to fits of lunacy. She had one of these during the treatment. The lens was removed in its capsule ; the resulting vision was $\frac{20}{100}$. She could read No. 1 Jaeger, but for a long time she insisted that she could not see at all.

FAULTY ENTRANCE OF THE KNIFE.

In another case, that of a lady of fifty-one, I introduced the knife, intending to make an upper section ; but I found its blade was pointed the wrong way, and finished with the lower. The patient made a good recovery—vision $\frac{20}{70}$,—she died a year and a half after the operation.

CASES IN WHICH SEVERAL MEMBERS OF A FAMILY HAVE HAD CATARACT.

I have operated upon a father and two sons for senile cataract. All the operations were successful, and they are all living. I have also operated on father and daughter, and a cousin of the father, in another family. I saw another cousin who had cataract, and a time was set for the operation, but he was attacked by acute disease and died. The mother of this cousin was also operated upon for cataract by a distinguished oculist.

In the family in which I operated upon the father and two sons, one son was fifty years of age at the time of the operation ; the other was thirty-six. In the patient of fifty the eyes were very prominent and myopic ; a small quantity of vitreous escaped ; the lens was removed in its capsule ; the eye recovered in eleven days; the resultant vision was $\frac{20}{50}$, which is still maintained some years afterward.

In the son of thirty-six, operated upon October 14, 1884, there was an attack of keratitis, but the vision reached was $\frac{20}{30}$, which lasted over a year, and after that was reduced by a membrane ; this again being a case where, although the lens was removed in its capsule, needling had to be performed. The needling again restored vision to $\frac{20}{40}$.

The father of these brothers, Dr. J. C., was seventy-six years of age at the time of this operation, Jan. 13, 1880, which was a Graefe, and is still living with good vision, as are his sons. They are Nos. 155, 171, 172, of the table.

In the other family the father was sixty at the time of the first operation, No. 144 of the table. I operated upon his fellow-eye successfully two years later, and he lived for fifteen years after. The daughter was but forty-four years of age when operated upon, Jan. 19, 1889.

Both operations were simple extractions, the former with Beer's knife, the latter with Graefe's knife, and two subsequent operations for membrane. Both, as well as the cousin of the father, had excellent results, No. 67 of the table. In this last case I extracted the lens in its capsule with an iridectomy. The patient was a lady of sixty-three, the operation was successful; the patient returned to the hospital with pain in her eye one month after she was dismissed. She was found to have iritis and pus in the anterior chamber. After the use of hot water every two hours, and atropine, the pain disappeared, and she was discharged with the same vision as she had when she left the hospital, $\frac{20}{70}$, and she continues to have it now, some five years afterward. I had, five or six years previously, treated her successfully for lachrymal catarrh by Bowman's method.

Two other cases, brothers, in whom I have now operated upon both eyes, are remarkable, because they are both under-sized men, with good mental capacity however, and microphthalmic. Their eyes, as to the cornea certainly, and apparently as to the eyeballs, are about half the size of ordinary adults', even of those below the medium size. The lenses bore no such disproportion in size, to those of ordinary eyes, as did the cornea. They were comparatively large. The vertical diameters of the second brother's corneæ is 6 *mm*, the horizontal only 7 *mm*. That of the older is not greater.

Priestly Smith[1] after examining 500 eyes, 250 males and 250 females, gives "the healthy cornea of the living eye of persons between five and ninety years of age an average horizontal diameter of 11.6 *mm*."

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[1] Transactions of the Ophthalmological Society of the United Kingdom.

The first brother, Floyd P., was twenty-eight years of age when I operated upon the right eye, Oct. 22, 1880, after vainly attempting to produce absorption by needling the lens; making no impression upon it, I removed the lens by Graefe's method, and good vision resulted; Nos. 32 and 34 of the table. Later, in November, I operated upon his fellow-eye, also successfully, and in February, 1889, upon his brother's eye, and finally, in January, 1891, upon his fellow-eye.

All of the operations have been successful, but all have required secondary operations except the last. The second brother had a diffuse opacity of the upper part of the cornea, which disappeared in ten days after, under the use of calomel. I am inclined to think that this opacity was due to the too prolonged use of cocaine, as he was very timid and insisted on having a great deal used.

REMOVAL OF LENS IN CAPSULE CONTRASTED WITH GRAEFE'S OPERATION.

A gentleman of seventy-five years of age, of Bennington, Vermont. This case is interesting because he had undergone a modified Graefe's operation on the left eye, which, after subsequent needling by another operator, had resulted in vision $\frac{20}{200}$. Floating bodies were in the vitreous humor, the patient was very much annoyed by them, and he desired to have another operation on the other eye, hoping to have none in that eye. A preliminary iridectomy was performed, the lens extracted in its capsule without accident, two weeks later. He had some senile delirium after the operation, and made a slow recovery. His vision was exactly the same as in the other eye, the floating bodies were equally troublesome, and continue so at this writing.

A lady, aged sixty-five, operated on July, 1881. This is of interest, because, like one just reported, I operated upon one eye by Graefe's method, and subsequently removed the lens of the other in its capsule. The eye first operated upon, after needling, had a resulting vision of $\frac{20}{70}$. The second, after a preliminary iridectomy, was removed in its capsule, October 27, 1886, and vision became $\frac{20}{40}$, and remains good.

There is another case in which one eye had been operated upon by another oculist by the Graefe method; the result was $\frac{20}{100}$ three years after. I removed the lens in its capsule, and six months after the vision was $\frac{20}{50}$.

I have observed that the flaps of the cases of extraction of the capsule heal much less readily than those of ordinary, simple extraction. This is probably due to the fact that the iris has been pushed a little out of place, when the lens emerges, and that this assists in causing the wound to gap. I think it important, where this operation is performed, that pains should be taken to replace the iris. As I have said, I have nearly abandoned the operation, because, while much more difficult than the simple extraction, the chief result which I hoped to secure—that is, the avoidance of pupillary membrane,—was not secured, and therefore it does not seem to me to have the advantage over the other operations. And yet, in one case--that of a distinguished writer of this country, given to the free use of alcohol,—I believe that the absence of the capsule as a source of inflammation, helped me to save the eye. There are certain cases in which the presence of the capsule is sure to excite tedious inflammation, which oftentimes ends in the permanent closure of the pupil. I believe that some, at least, of the cases that figure in reports published in a few months after the cases were operated upon, finally become cases of closed pupil from iritic and capsulary inflammations. In cases apparently predisposed to inflammations of the iris and capsule, the removal of the capsule is a very great advantage. Neither do I think that the trifling loss of vitreous, which is more apt to occur in an extraction in the capsule, by my method, is particularly prejudicial to a good result. A slight or even a considerable loss of vitreous does not, so far as my experience goes, predispose an eye to subsequent detachment of the retina or hyalitis, as is sometimes said.

LOSS OF ONE EYE FROM INFECTION BY LACHRYMAL CATARRH; FELLOW-EYE SUCCESSFULLY OPERATED UPON.

CASE 169 of the series. Here a man of seventy-one was operated upon, October 12, 1881, under ether. The wound was infected by lachrymal catarrh, and after it had closed it reopened. The result was a closed pupil and perception of light. Six months after I removed the lens of the fellow-eye in its capsule, without, however, setting out to do so until the section was made and it was seen that the lens was ready to escape. The vision finally became $\frac{20}{50}$.

BAD ECCENTRIC VISUAL FIELD ; GOOD RESULT.

In one case, No. 80, a woman of fifty-two, before the operation the peripheric visual field was very poor. The central perception of light was, however, good, and the result was $\frac{20}{100}$ in the central field. The ophthalmoscope showed extensive choroiditis disseminata.

HEMORRHAGE FROM THE IRIS.

Case No. 83 of table, a man of fifty-nine years of age, had a sad interest, because hemorrhage from the iris occurred after the operation upon the first eye, and finally the sight was lost. The operation on the other eye was undertaken four years later, with the precaution of preliminary iridectomy. All went well for ten days, when a hemorrhage occurred, and that gradually became absorbed. The patient secured a very moderate amount of vision, $\frac{2}{200}$, which he still maintains.

RE-OPENING OF A WOUND ; SEPTIC PNEUMONIA ; DEATH.

A man of sixty-five, June 2, 1888, an old soldier, who had had an operation upon the bones of the right forearm. The wound was open, but filling up by granulation. His attending surgeon considered him in as sound a condition as he would ever be. He feared nothing from this wound ; but a good operation, removal of the lens in its capsule, was followed by the re-opening of his wound, with severe inflammatory action and erysipelas. The eye, however, during his illness healed perfectly, and he had good sight. It could not be positively measured, on account of his severe illness—it was certainly $\frac{20}{100}$; but he died of septic pneumonia, one month and ten days after the cataract operation. The fellow-eye had been lost by inflammation.

In four cases I have operated, in spite of the existence of chronic conjunctivitis, which, however, I had treated, for some weeks before, without, however, having subdued the inflammation entirely. In all these the result was good.

In the case of John S., aged seventy-five, No. 101, the lower section was made ; it was encroached upon after partial healing by the lower lid, and suppuration of the edge of the wound occurred, although a very moderate amount of vision was left. I think the liability to this accident is a great objection to a lower section.

I have found that not only senile delirium, but also malaria is apt to develop in persons undergoing cataract operation, or at least may develop, and I have seen benefit in malarial subjects, in keeping the patient constantly under the influence of quinine in moderate doses.

The operation of the removal of the lens in its capsule is generally followed by considerable astigmatism—more than usual in simple extraction. Among the poorer patients it was impossible to induce them to wear the proper correcting glasses. Some of my results would have been better if they had been measured with the full correction of the astigmatism.

PROLAPSE OF THE VITREOUS SOME SIX DAYS AFTER THE OPERATION ; SYMPATHETIC OPHTHALMIA ; LOSS OF BOTH EYES.

The case of T. M., aged sixty-five. Operation at Manhattan Eye and Ear Hospital, April 15, 1890, is one of sad interest. The eye was doing perfectly well four days after the operation, when he snapped his eyelids together, just after the lids had been washed, prolapsed the vitreous humor, and the eye went on to total destruction. Although the eye was promptly removed, sympathetic irritation occurred in the other, and that also was lost, in spite of earnest efforts, removal of the lens, iridotomy to secure something from the wreck, and so forth.

I think the matter of the pre-existence of myopia is not always fully appreciated in estimating as to the visual power after an operation. Schweigger has called attention to this subject ; certainly statistics taken from a number of myopic patients would not give apparently as good results as those from hyperopes.

MYOPIA ; PARTIAL SUCCESS ; FIVE SUBSEQUENT OPERATIONS.

One of the series illustrating this and other points is that of a gentleman from Buffalo, of sixty-eight years, who was first operated upon by me on Feb. 10, 1879, having lost the left eye by an unsuccessful operation in the hands of another surgeon. From all accounts he never had more than $\frac{20}{100}$ distant vision with a concave glass. A modified Graefe was performed and his vision was only $\frac{8}{200}$, but he got about very well, and read large type. The pupil closed. He has had 2 needlings, 2 iridectomies, and 3 iridotomies, and in October, 1890, two years after the last operation, had $\frac{10}{200}$. Loring's operation for iridotomy was successful in his case in restoring considerable vision, when the sight was reduced to perception of light, and one competent ophthalmologist thought all chance of an improvement was gone. The patient is still living, at eighty years and upward, and has to thank his own pluck for the modicum of vision that remains to him.

One of the cases in the table was reported at a meeting of the American Ophthalmological Society, 1863, p. 69. It is one of the Liebreich operations. As I have known it to happen in another case, and in the hands of an experienced operator, the lens was dislocated, instead of being removed; but in my case, fortunately, the wound was opened one week later, the dislocated lens seized with a cystotome, and readily removed without accident, and good vision resulted.

VOMITING.

In one case in which ether was given, the patient vomited before the bandage was applied. She was very unruly, and complete prolapse of the iris occurred. The flap was bent over—it was very difficult to replace it. Suppuration followed. In another case where an excellent result was obtained, vomiting was very profuse twice after the bandage was applied, yet no harm resulted.

There were six eyes, two besides the four of the Powell family, that could properly be called microphthalmic, they were so small, yet the results were as favorable as in eyes of the proper size, although the extractions were rather more difficult.

In three cases, the patient, after having lost one eye at my hands, has consented to an operation on the other eye, and has obtained useful vision.

I remember one unfortunate case, however, not included in this table, because I have never been able to find the records, of a patient who lost one eye from suppuration of the iris, and when the wound had closed some months after, I operated on the fellow-eye with the same sad result, and yet nothing was at fault with the technique, the wounds healed very kindly, and as in case No. 186 suppurative iritis supervened. In this latter case, I have always felt that it began with a chill, induced by a change from a sunny room to one without sunlight, and possibly infected with sewer gas. Fortunately for the patient, a previous operation upon the fellow-eye had been successful.

LINEAR EXTRACTION.

The linear extractions deserve mention:

Fourteen eyes in all were operated upon by this method; there was only one failure. They were usually begun by an incision with an iridectomy knife, not always accompanied by an iridectomy, but always when there was difficulty in removing the whole of the lens matter. Most of the cases are found in this class, but five eyes in three persons, all males, were idiopathic. When I saw them they were fully developed, and it was impossible to say positively whether or not they were congenital. In one case, that of a Catholic clergyman of 31, on whom I operated by discission in March, 1887, with a resulting vision of $\frac{20}{40}$. In 1890, when the cataract in the fellow-eye became ripe, I removed it by linear section, and saved the patient much time. The operation did well, but his vision was not, when I saw him last, two months after the operation, as good as the other, $\frac{20}{70}$. In another case, that of a dentist of Kingston, N. Y., a linear section with iridectomy, resulted in v. $\frac{20}{20}$. Case 183 of table. In a third case I operated upon both eyes, by linear section, a young man, a clerk of 27; the first in March 1887, on the fellow-eye, the right, in May, 1888. This patient gave a clear history of injuries to his head, to which he attributed the loss of sight. He had an injury from a sand-bag one year before he consulted me, and when a small boy he was hit in the forehead by a stone. He got good vision in each eye, and uses his sight all day as an invoice clerk.

The risks of a linear section are very slight as compared with those of a flap, as is well known. I think the field of its employment might be profitably enlarged. It is well adapted to many cases of cataract beside the traumatic form, for which I have, like others, usually employed it.

I have included two cases of extraction of cataract in my account, in neither of which was it expected that vision would result. In one, the operation was performed for cosmetic reasons. The patient was very myopic, had prominent eyelids, and the deformity from the cataract was considerable. A linear incision was made, and after evacuating a small portion by pressure and counter-pressure, a very large lens was slowly extracted by the use of Panas' syringe. I think this is valuable not only for washing out the soft cortical matter, but also soft cataracts. It is a much better instrument than the old suction instrument of Teale.

This analysis might be continued in greater detail, but it has already perhaps exceeded reasonable limits. To conclude, from my present standpoint, I prefer simple extraction, of course with a narrow knife, closure of the eye with a bandage, which may be changed every twenty-four hours, if needful for the comfort of the patient, without opening the eye, after the first twelve hours. I do not insist upon a rigid decubitus; I usually instil a drop of a 1:500 solution of sulphate of eserine after the operation. I think it very important to get the cortical matter away, as do all operators I believe. For this purpose, I think Panas' syringe valuable, although as yet I have used it very little. Panas himself has given up injections of the anterior chamber with mercurial solutions, and uses a solution of boric acid. Preliminary iridectomy, with Forster's massage, is a procedure to be advised, in my opinion, in all cases where we have reason to fear, either from difficulty in the operation and from the condition of the eye, that it will be difficult to extract the lens without accident. If after making the section, it is seen that the lens will readily escape without a capsulotomy, its removal in its capsule is to be recommended. When subsequent capsulotomy is necessary I prefer Galezowski's knife; when I am obliged to perform an iridotomy, Loring's method, that with the use of a narrow Graefe's knife, seems to me the best.

ON METASTATIC CARCINOMA OF THE EYEBALL.

By Dr. JOHANN MITVALSKY.

ASSISTANT TO THE BOHEMIAN EYE-CLINIC IN PRAGUE.

Translated by Dr. Ward A. Holden, New York.

(With six drawings on Plate xvii. of vol. xxi., Germ. ed.)

THE rarity of metastatic carcinoma of the eyeball, six cases only having been described, leads me to report two analogous cases observed in the eye-clinic at Prague, and to speak of some particular points in the histories which are common to all these cases.

The cases hitherto reported are as follows:

The first case, described by Perls,[1] was a metastatic carcinoma of the choroid in both eyes; an anatomical examination only was made; the lung was the seat of the primary tumor.[2]

The second case, described by Hirschberg,[3] was a metastatic carcinoma of the choroid in both eyes; a clinical examination only was made; the right mammary gland was the seat of the primary tumor.

The third case, described clinically by Schöler,[4] anatomically by Uhthoff,[5] was a metastatic carcinoma of the choroid in both eyes, which involved sclera, papilla, and optic nerve, presenting a tumor in the posterior segment of the eyeball; the left mammary gland was the seat of the primary growth.

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[1] *Virchow's Archiv*, vol. lvi.

[2] Abstracts of Cases 1, 2, 3, 4, and 6 are to be found in a paper by Schultze, Arch. of Ophth., 1890, p. 228.

[3] *Centralbl. f. prakt. Augenheilk.*, p 376, 1882.

[4] *Berliner klin. Wochenschrift*, No. 7, 1883; and *Centralbl. f. prakt. Augenheilk.*, p. 236, 1883.

[5] *Ibid.*, p. 412, and Report of XV. Meeting of Heidelberg Ophth. Soc., p. 204.

The fourth case, described by Hirschberg and Birnbacher,[1] was a metastatic carcinoma of the choroid in the left eye, examined both clinically and anatomically; the primary growth was located in the right mammary gland.

The fifth case, described by Manz,[2] was a metastatic carcinoma of the choroid in both eyes, only examined clinically; the right mamma was the seat of the primary disease.

A woman, æt. 50, noticed a mammary tumor early in 1883, which was extirpated in August of the same year. Toward the end of October she noticed a cloudiness before the left eye and later before the right. Examination, middle of October, R, V$=\frac{1}{9}$; L, V= fingers at 3 feet, T not increased (L, rather diminished finally). Both eyes showed extensive detachment of the retina. Vision was finally completely lost. Patient died in December.

The sixth case, described by Schapringer,[3] was a metastatic carcinoma of the choroid in the left eye, examined clinically and anatomically; the primary growth being in the right mammary gland.[4]

The ninth case, described by Schultze,[5] was a metastatic carcinoma of the choroid in the left eye, examined clinically and microscopically; the primary growth being in the left mammary gland.

Mrs. X., æt. 34, showed a detachment of the upper outer portion of the left retina. The enucleated ball showed two distinct flat tumors. One year before, a carcinoma of the right breast was discovered and extirpated. Patient died of bulbar paralysis a few months after the ocular symptoms came on.

The tenth case, described by Ewing,[6] was a metastatic carcinoma of the ciliary body and iris in the left eye, examined clinically and anatomically; the primary growth being in the left breast.

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[1] *Graefe's Archiv*, vol. xxx., p. 113.

[2] *Ibid.*, vol. xxxi., p. 101.

[3] Schapringer, *Amer. Journ. of Ophth.*, vol. v., No 10.

[4] Since this paper was originally published, three further cases of carcinomatous metastases in the eyeball have been reported. Abstracts of these are introduced here, as Cases 9, 10, and 11.—TRANSLATOR.

[5] Schultze, these ARCHIVES, 1890, p. 227.

[6] Ewing, *Graefe's Archiv*, 1890, p. 121.

Miss B., æt. 32, complained of failing vision for three months, and showed detachment of the retina with increased tension. Eye enucleated. At this time a carcinoma as large as an apple was found in the left mammary gland.

The patient's general condition was bad and she vomited frequently.

The eleventh case, described by Wadsworth,[1] was a metastatic carcinoma of the choroid in the right eye, examined clinically and anatomically; the original disease being located in the right breast.

Miss T., æt. 46, complained of failing sight. The retina was pushed forward by a tumor, and detached peripherically. Tension was not increased. The enucleated ball exhibited a shell-shaped carcinoma with areas of hyaline degeneration. A moderately-sized carcinoma had been removed from the right breast sixteen months before.

Gayet[2] has recently described a case of metastatic tumor of the choroid, which appeared as an adenoma following an adeno-carcinomatous growth in the stomach. Since this interesting case was not one of carcinoma in the strict sense of the word, it will be passed over.

THE TWO NEW CASES.

The seventh case of metastatic carcinoma of the choroid, in the left eye, the primary growth being situated in the left mammary gland.

Franziska Kucera, æt. 46, consulted Prof. Schöbl, Feb. 16, 1886, complaining that the sight of the left eye had been failing for 2 weeks. A carcinoma had been removed from her left breast in 1884. After four months there was a local recurrence of the growth, with swelling of the axillary and supraclavicular lymphatic glands. A second operation was made, since which her condition has been good. No history of hereditary disposition. Examination of the somewhat emaciated patient showed no nodules in the cicatrix in the breast, but a number of enlarged hard lymphatic glands in the axillary and supraclavicular regions were found.

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[1] Wadsworth, Transactions of Amer. Ophth. Society, 1890.

[2] *Archives d'Opht.*, Mai-Juin, 1889.

The right eye was quite normal, and the left appeared so exteriorly; L, V = $\frac{6}{120}$ with direct fixation. With the ophthalmoscope the media appeared clear. A vertically oval growth of three disc diameters extended laterally from the papilla to the macula lutea. It had a dirty yellow color, with isolated pigmented points. Its central position showed an elevation of three or four dioptries; its periphery passed gradually over into the emmetropic fundus.

The central portion of the growth exhibited small vessels of irregular course, which were not in connection with the retinal vessels and were therefore probably of new formation. T normal. Perimetric examination revealed a scotoma corresponding to the growth.

A diagnosis of metastatic carcinoma of the choroid was made.

March 1, 1886, the ophthalmoscope showed movable thread-like opacities in the vitreous, and a detachment of the retina involving two thirds of the fundus. T diminished.

March 15th, intolerable pain in eye, forehead, and head; retina totally detached. Ball glaucomatous; enucleation performed. Two months later the patient died with symptoms of metastases in the lungs and brain. The right eye remained unaffected.

The enucleated ball was hardened in a mixture of Müller's fluid and picric acid, and divided by a horizontal section. The antero-posterior diameter was 24.5 *mm*, the æquatorial 22.5 *mm* (Fig. 1). The inner third of the choroid is macroscopically normal, its outer two-thirds are replaced by a shell-shaped mass of dirty yellowish-gray color, which extends beyond the ora serrata on the temporal side, nearly reaching the ciliary body. The greatest thickness of the mass is 2 *mm*.

The growth surrounds the papilla without altering it macroscopically. The inner surface of the tumor exhibits shallow depressions, and the surface toward the sclera has a slightly wavy contour. The tumor consists of a dense framework and a brittle intervening substance. The retina shows a funnel-shaped detachment. Only the anterior portion of the choroid, particularly inward and upward, remains free from the growth.

After imbedding in celloidin, total sections of the ball were made, and also flat sections of the tumor. All the preparations show numerous yellow spots, varying from the size of a pin-point up to that of a hemp-seed, which do not stain with the rest of the tumor, and which drop out as plug-like masses when the preparations are washed.

Microscopically the tumor presents the appearance of a carcinoma, which, as the relative proportion of connective-tissue framework and nests of epithelial cells varies in different portions, is to be considered in part a scirrhus, in part a simple carcinoma (Figs. 2 and 3). In general the central portion of the tumor, in transverse section, shows rather the character of a fibrous carcinoma, with a predominating dense connective-tissue framework, and relatively few small and isolated cell nests; the peripheral portions, however, show a more delicate connective-tissue framework, with large meshes which are filled with cell nests.

The connective-tissue framework exhibits everywhere choroidal pigment, indicating that its origin must have been in the pigmented choroidal stroma. This framework has a markedly fibrillar structure, and contains a few oval or spindle-shaped nuclei of connective-tissue cells, and a few leucocytes near the scleral surface.

The carcinoma cell nests are of all shapes and sizes (Figs. 2 and 3) and the tubules have been cut in every direction.

The yellow unstained plugs, previously mentioned, would seem to consist mostly of degenerated cells from the interior of the nests, with some pigment, and nuclei of connective-tissue cells, indicating some participation of the framework of the tumor in the process.

The principal change in those portions of the choroid which are being invaded by the carcinomatous tubules is an increase in volume; the pigment cells and the connective-tissue fibrillæ are increased in number, producing a thickening of the choroid, which is again increased by the obstruction of the circulation. Near the nasal margin of the tumor the meshes of the suprachorioidea are dilated by the remains of old hemorrhages. The choroidal veins are dilated and many of the arteries are small. There is no marked infiltration of round cells about the vessels. The tumor here spreads chiefly in the suprachorioidea, and the neighboring vessels are dilated and filled with degenerated blood corpuscles.

At the temporal margin (Fig. 3), however, the carcinoma is seen to be developing mostly in the ground-substance of the choroid, below the chorio-capillaris. The vessels are moderately dilated. The suprachoroidal space is enlarged and is the seat of an extensive hemorrhage (Fig. 3 *b*). Some preparations show carcinomatous nests within the hemorrhage, either connected with other tubules or isolated (Fig. 5). These evidently represent carcinomatous processes growing through the hemorrhage. The lamina vitrea is everywhere preserved and covers the inner surface of the tumor, following its elevations and depressions. The inner surface of the lamina vitrea is covered with pigment epithelium, raised here and there by a homogeneous exudate. The epithelial cells have lost their pigment for the most part and are undergoing a hydropic degeneration.

The retina is well preserved for the most part, showing, however, a dilatation of its blood-vessels, a displacement of the rods and cones, and a cystoid degeneration peripherically.

The papilla has not become involved in the growth, the vitreous shows a fibrous contraction, the lens is normal. The iris and ciliary body are unchanged, the chamber angles are open, the anterior chamber is filled with a fibrinous exudate. The cornea is unchanged, the sclera shows a round-celled infiltration in places, the bulbar conjunctiva is thickened and infiltrated with round cells, particularly about the vessels.

REMARKS ON THE ANATOMICAL CONDITION OF THE BALL IN OUR FIRST CASE.

While in its clinical course our case differs in no important particulars from those previously described, it shows one histological change, which has not been noted in the other cases, viz., the presence of a number of degenerated foci, which have been described above. At first glance these degenerated foci seemed to be the necroses which are often found in old carcinomata, but, studied more closely, their peculiarities and their relations to the surrounding parts seem to render this view improbable. Let us first see whether these degenerated parts have arisen in the same manner as those which we are accustomed to see in carcinomatous tumors, and which arise, as is known, through retrograde changes, such as mucoid, hydropic, and fatty degeneration.

Were this the case, we should be able to find all the successive stages of the metamorphosis. Under a high power, however, no carcinoma cells are found which exhibit signs of any of these degenerations, although had the necrosis been caused in this manner, the cells about the necrotic foci should exhibit some changes of this sort.

This is another point also which speaks against this origin of the necrotic foci. It is evident that, in an extensive carcinomatous necrosis, the carcinoma tubules, as well as the connective-tissue framework which supports them, might be destroyed, and it would not have surprised us to have found a quantity of choroid pigment within such a necrotic mass. But the case is different when a small necrotic focus crowded with pigment is discovered within a normally formed and well-preserved carcinoma tubule, while the tubule itself shows no trace of pigment. Here we are compelled to acknowledge that the necrotic portion could not have arisen from the surrounding carcinoma cells, since in such a case it must have been free from pigment.

If a preparation of the tumor be observed through a magnifying glass, the large hemorrhages which were noticed particularly in the suprachoroidal space at the temporal margin of the tumor (Fig. 3 *b*), are seen to resemble the necrotic foci both in color and in general appearance. Microscopically, also, the hemorrhages have the same yellow color as the necrotic foci.

As has been said, the hemorrhages are only well preserved in their anterior portions; posteriorly toward the developing carcinoma, the hemorrhages with their fibrinous networks degenerate into a finely granular mass, in which may be seen leucocytes, swollen cells of the connective-tissue tracts, and pigment epithelial cells which have lost most of their pigment. Between these, clumps of free-lying choroidal pigment are seen. The carcinoma cells are seen spreading toward the hemorrhages, following the connective-tissue septa (Fig. 3 *c*), and even in the hemorrhages themselves we see carcinoma tubules, either connected with the neighboring tubules or already separated from them (Fig. 5). When it is seen that these hemorrhages exist also on the nasal side, and that from the obstruction of the circulation the conditions were not favorable to their resorption, what else can one suppose but that these hemorrhages, instead of being absorbed, coagulated into unformed masses, which eventually became surrounded by the developing carcinoma cells? The central or more eccentric final position of the mass in its sheath of carcinoma cells, depends upon the further development and increase of these latter. If now a portion contained within a small mesh be surrounded by the carcinoma cells, a small apparently necrotic focus results, which may be entirely colorless, or which may contain a greater or less quantity of choroidal pigment. When larger portions are involved, some tracts of connective tissue are also included, and these degenerate for the most part, and are represented only by clear stripes and tracts of pigment. In this manner the connective tissue of the focus is separated from the surrounding tissue, and but a trace of it is found here and there in a sequestered portion.

A number of other facts point toward such a manner of development for the necrotic foci, and the existence of all of them may be easily explained in this way. The development of the very small foci is probably as follows: the cells of the carcinoma tubule endeavor to spread into the interior of a necrotic focus following a bundle of connective tissue, but the carcinoma cells are only able to surround some small portions in the smallest peripheric meshes, and these being cut off become separate small foci.

Some of these small foci may arise in another manner. The clear wall of some of these contains nucleated spindle-cells, and the shrunken, yellow, necrotic contents are seen to be separated from the wall, leading one to believe that this had been a capillary, whose contents had undergone a granular degeneration. We may suppose that in consequence of the obstruction to the circulation which the tumor caused, a number of dilated capillaries were cut off from the general circulation and the blood contained within them coagulated.

If the periphery of the tumor be examined, three stages of the process by which the carcinoma cells invade the necrotic foci may be followed. The freshest foci of the periphery, partly surrounded by carcinoma cells, show a number of scattered darkly stained nuclei, but these are not more numerous near the sheath of carcinoma cells. Later the peripheric zones of the focus, near the sheath, show a considerable increase in nuclei, evidently from the neighboring carcinoma cells, but the necrotic focus is still closely connected with its sheath of carcinoma cells. In the latest stage these nuclei are rarely found, but the necrotic focus is separated from its sheath by a free interval. The first stage is that in which the cells develop in one direction, forming a tubule; the second that in which the tubule ceases to develop longitudinally, but becomes thicker; the third that in which the necrotic focus becomes surrounded and appears cut off from the living tissues, like a sequestrum.

There is a further peculiar appearance to be spoken of. In the periphery of the tumor, in the centre of large nests of carcinoma cells, there are found parts which consist only of these darkly stained nuclei, held together by a yellow homogeneous cement-substance (Fig. 6 *a*). This is, however, nothing else than the peripheral portions of necrotic foci surrounded by carcinoma cells in the first and second stages, cut tangentially.

Let us consider now the development of the tumor. The carcinomatous metastasis has occurred in the central portion of the choroid, from which it has spread in all directions, so that the two temporal venæ vorticosæ have soon been surrounded, and the circulation through them blocked. This has caused a marked dilatation of the vessels, and the veins have burst from the distention, permitting the escape of blood, or their walls have been eroded by the carcinoma cells, so that repeated hemorrhages have occurred in the periphery of the tumor. But since the conditions for resorption were unfavorable the hemorrhages have undergone a granular degeneration, and have coagulated later into masses, whereby the tumor has been forced to change its previous structural character of a simple carcinoma, since the cells have been compelled to spread along the connective tissue surrounding or pervading the coagulated masses, and the cellular sheaths described above have been formed.

If the presence of crystallized blood pigment could have been demonstrated in the coagulated masses, our hypothesis as to their origin would have been proven. A negative result, however, would not speak against our hypothesis, since the specimen had been hardened in Müller's fluid and picric acid, both of which agents render the test of Quincke with ammonium sulphide, and Perls' iron reaction with potassium ferro-cyanide, very uncertain or negative. And in fact both the tests resulted negatively in this case, but neither were they positive in the case of the degenerated corpuscles in a large unquestionable hemorrhage near the temporal margin of the tumor.

The eighth case was a metastatic carcinoma of the choroid in the left eye, a clinical examination only having been made; the seat of the primary tumor was the left breast.

Skrivan, Franz, æt. 35, came to our clinic October 25, 1887, complaining of failing vision in the left eye. He had always been healthy until October, 1886, when he noticed a hard lump near the left nipple, which developed in a relatively short time into a large tumor, which was diagnosed carcinoma, and was removed, together with the axillary glands involved, in March, 1887. Microscopically the tumor presented the usual appearance of a scirrhus.

Status præsens.—Axillary and supraclavicular glands enlarged. Right eye normal; in the left eye the media are clear, but an irregular vertically oval, elevated tumor is seen, extending from the macula lutea some four disc diameters toward the temporal side, of a dark yellow, dirty color, with white patches here and there, and occasional pigment accumulations. Its greatest elevation is four dioptries. T normal. $V = \frac{6}{60}$, with eccentric fixation.

The growth developed rapidly; opacities of the vitreous appeared; and the retina became detached. Metastatic tumors developed in the cranial periosteum, and the patient died with cerebral symptoms in December, 1887.

GENERAL REMARKS IN REGARD TO THE CASES OF METASTATIC CARCINOMA OF THE EYEBALL PREVIOUSLY REPORTED.

Eight[1] cases of pure carcinomatous metastasis have been observed in the eyeball, five of which (those of Perls, Schöler-Uhthoff, Hirschberg, Birnbacher, Schapringer, and our first case) were examined anatomically; while the other three (those of Hirschberg, Manz, and our second case) were only studied clinically.

The seat of the primary growth was the mammary gland in seven cases, 87.5 per cent., and in but one case, 12.5 per cent., did it show another location—that of the pleura in Perls' case.

The age of the individuals affected varied, the youngest being 28, three being between 30 and 40, two between 40 and 50, and the other two over 50, the eldest being 52. Six cases occurred in females, two in males.

It is not surprising that the ocular metastases were always in the choroid, since the detached carcinoma cells would be much more likely to enter one of the twenty short ciliary arteries than the single central retinal artery, whose lumen is almost equal to that of the ciliary arteries. It is, however, possible for a metastatic carcinoma to develop in the retina, since Schiess-Gemuseus has reported a case of sarcoma which had been followed by metastases in the papilla and the neighboring portion of the retina.

Since the germs of the metastasis are mostly single cells, the first beginning of the metastases must take place in those layers of the choroid which have the smallest vessels, viz., Sattler's layer and the chorio-capillaris, and the first development probably occurs within the lumen of the vessel; soon, however, the choroidal stroma thickens and forms the framework for the carcinoma. Schöler-Uhthoff say that carcinoma cells are distinctly seen within the lumen of the vessels; Hirschberg-Birnbacher say, however, that no carcinoma cells were found within the vessels in their case. In my case, single nests of carcinoma cells seemed surrounded by typically distended vessels walls.

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[1] Eleven cases have now been reported.

The tumors may be either shell-shaped or tuberous in form, and we may employ the terminology which Fuchs[1] has suggested for the uveal sarcomata, calling the former diffuse and the latter circumscribed. Seven of the cases described belong to the diffuse variety, and one, that of Hirschberg-Birnbacher, was distinctly tuberous.

Histologically the metastatic growth corresponded to the primary one as a rule, the type being usually that of a carcinoma simplex or a scirrhus.

In half the cases only the left eye was affected, the primary growth being in two cases in the left mammary gland and in two cases in the right. In the other four cases the metastases were bilateral, both eyes becoming affected simultaneously, or one shortly after the other, the metatastic process being probably the same in both.

The intra-ocular tension varies greatly in these cases. Hirschberg found the tension diminished; in Schapringer's case and our second case it was normal; Manz found it not diminished; in the Schöler-Uhthoff case and in our first case a consecutive glaucoma developed. The condition of the tension then throws no light on the diagnosis. In the present condition of our knowledge the variation cannot be satisfactorily explained.

The clinical course of the disease should be spoken of. The first beginning of a metastatic carcinoma is rarely seen. Hirschberg, in his case, found the media clear, several small yellow foci being seated close to the papilla, and in the other eye a larger diffuse thickening of the choroid was seen, which elevated the retina 1.5 *mm.* The tumors grew rapidly and the retina soon became detached. In all the cases, where the case was seen at an early stage, the periphery of the fundus was normal. Later, opacities of the vitreous appeared, and the retina became detached downward and finally totally. Schöler noticed a considerable detachment of the retina, which at a later examination, shortly before the death of the patient, had disappeared.

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[1] Fuchs: "Das Sarcom des Uvealtractus," p. 115, Vienna, 1882.

In some of the cases in which the tumor was shell-shaped the retina was pushed forward, causing a more hyperopic refractive condition of the eye. In but one case (Schöler-Uhthoff), was there an extra-bulbar development of the tumor, which was revealed first by a papillitis. All the cases showed a rapid growth of the tumor.

It is interesting to notice the interval which elapsed between the first appearance of the primary tumor and the development of the metastasis, as well as that which elapsed between the first appearance of the metastasis and the fatal ending.

In Hirschberg's first case the primary growth had developed for nine years before the first ocular symptoms appeared, after which death followed in a year. In the Schöler-Uhthoff case, the primary tumor lasted six years before the vision began to fail, after which death followed within a year. In the Hirschberg-Birnbacher case, the length of these intervals is not expressly stated; the latter interval would seem however to have been but a few months. In Manz's case the metastasis came on nine months after the discovery of the mammary tumor, and death followed some months later. In Schapringer's case the metastasis appeared two years after the amputation of the first, and death occurred four months later. In our first case two years elapsed before the eye became affected, and the patient died three months later. In our second case the ocular symptoms appeared somewhat more than a year after the discovery of the mammary tumor, and the patient died three months later.

It is thus seen that the metastases in the eye came on in the same manner as do those in other parts of the body, that like the latter they appear at a varying interval after the development of the primary tumor, and that like the other metastases they are a sign of the approaching end, which in the cases known came after an interval varying from three months to a year.

The discovery of a metastatic carcinoma in the eye may be of practical interest in fixing the date of the lethal ending, and also in diagnosis when metastases of internal organs cannot be discovered by physical means. It might also be of value in enabling us to differentiate between an inflammatory and a neoplastic disease—*e. g.*, of the pleura or pericardium.

The diagnosis is not difficult.

The therapy consists in combating the symptoms, and enucleation should be done only when the pain of a consecutive glaucoma is not relieved by an iridectomy or sclerotomy.

Explanation of the Figures.

FIG. 1.—Section of eyeball, natural size.

FIG. 2.—A portion of the tumor slightly magnified. *a.* Necrotic foci with no trace of choroidal pigment. *b.* Necrotic foci with tracts of pigment.

FIG. 3.—Section through the temporal margin of the tumor. *a.* Necrotic focus with scattered nuclei. *b.* Large hemorrhage in the supra-choroidal space. *c.* Tracts of carcinoma cells developing along the course of connective-tissue septa, toward the hemorrhage. *d.* Lamina vitrea with the pigment epithelium.

FIG. 4.—A necrotic focus surrounded by a crescentic sheath of carcinoma cells, highly magnified. The difference between the two sorts of nuclei is distinctly shown only in the periphery of the focus.

FIG. 5.—An isolated group of fresh carcinoma cells, situated in the middle of the large supra-choroidal hemorrhage, adherent to a connective-tissue bundle.

FIG. 6.—Section through a carcinoma tubule, in which the periphery of a necrotic focus has been touched, recognizable by the group of darkly stained nuclei (*a*).

A CASE OF PERIVASCULITIS RETINÆ.

(REPORTED FROM THE OPHTHALMIC INSTITUTE AT WIESBADEN.)

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Translated by Dr. Ward A. Holden, New York.

(*With two illustrations in the text.*)

AFFECTIONS of the uveal tract and of the retina after the age of puberty are more rarely observed than parenchymatous keratitis as the first and isolated manifestations of hereditary syphilis; yet they also undoubtedly occur.

At the suggestion of my chief, Prof. H. Pagenstecher, I publish the following hitherto undescribed case, in which hereditary syphilis manifested itself for the first time in a young man of eighteen, who until then had been entirely well, by extensive changes in the veins of both retinas. The case may possess some points of interest for my colleagues.

H. B., æt. 18, presented himself at the Ophthalmic Institute, Feb. 20, 1888. He declared that twelve days ago on rising he suddenly experienced a peculiar sensation of heaviness and pressure in the right eye. He at once closed the left, and discovered to his horror marked obscuration of the previously entirely healthy right eye, the cause of which he did not know. The obscuration, he stated, had decidedly increased during the last few days.

Stat. praes.: R E, $V = \frac{6}{24}$; L E, $V = \frac{6}{6}$; cornea, iris, refractive media of the right eye perfectly normal. The ophthalmoscopic examination in the inverted image shows marked capillary injection of the OD, with diminution of the transparency, especially at the periphery, which is slightly hazy. In the tissue of the OD there are two delicate streaky hemorrhages along the vena papillaris inferior ; the latter is about half as broad again as usual, and of a decidedly dark color. The other vessels of the OD are normal. When the vena papill. inf. is traced beyond the edge of the OD, it is seen to project beyond the level of the retina as a blackish-red cord of double the normal width. The width and dark coloration are greatest about 1 PD from the OD. After division into the v. nasal. and tempor. inf. both these veins, especially the latter, appear broadened and tortuous, even to their most delicate ramifications, and edged in their whole course with fresh, brownish-red hemorrhages, which near the OD present a radiate, streaky appearance, while at the periphery they are of an irregular shape, and often entirely conceal the blood-vessel. The v. nasalis and tempor. sup. show similar conditions, though less marked, so that the whole periphery of the fundus appears covered with fresh hemorrhages. The largest is at the macula lutea, an irregularly oval brownish-red patch of about 1½ PD, with sharp edges, giving rise to a positive, central, incomplete scotoma. The perimeter also shows several smaller peripheral incomplete scotomas, corresponding to the largest hemorrhages. About 1½ PD downward and inward from the macula an evenly thick, white cord, partly covered with hemorrhages, is visible, which joins the curiously tortuous v. tempor. super.

The tissue of the retina between the veins, where there are no hemorrhages, is perfectly transparent. The calibre and walls of the blood-vessels are quite normal.

The only pathological condition in the left eye is a circumscribed change in the wall of the v. nasal. sup., about 2½ PD from the OD. The normal red here shows a slight whitish discoloration, which seems to be formed by the confluence of grayish-white scales, between which reddish dots shine through here and there. The calibre and course of the vein on both sides of this spot are perfectly normal.

The right eye therefore showed the picture of hemorrhagic retinitis, due to a local affection of the blood-vessels, as shown by the changes in the v. temp. super. At the same time I suspected that the slight discoloration in the wall of the v. nasal. sup. of the left eye might be the beginning of a similar affection.

A thorough examination of the powerfully built young man showed that the other organs, especially the kidneys and heart, the pulse wave, and condition of the blood, were entirely normal. On the penis nothing could be found to indicate syphilis ; infection was positively denied by the patient, whose veracity could not be doubted. There was, however, marked induration of the cervical, cubital, and inguinal glands, so that in view of the changes in the eye the diagnosis of probably hereditary syphilis was made. Questions in this direction, however, were at first unsatisfactory. The patient was at once subjected to inunction (5.0 ungt. hydrarg. pro die).

On Feb. 28th, after thirty-five grms. had been rubbed in, his condition was decidedly worse.

In the right eye there was still $V = \frac{6}{24}$. But the ophthalmoscopic picture more and more approached that of incomplete thrombosis of the central vein. The OD looked " like the head of a Gorgo surrounded with dark locks " (Becker). In every direction the distended black veins rose far beyond the plane of the retina, often surrounded by perfect pools of blood, so that the thought naturally arose that a similar condition had developed in the central vein itself. The hemorrhage at the macula remained unchanged. Judging from the ophthalmoscopic appearances, at least two thirds of the retina were occupied by hemorrhages.

The most marked changes had taken place in the left eye during the week (*cfr.* Fig. 1). Color of the OD more reddish than normal and slightly hazy, especially in the nasal half. Outlines of outer edge indistinct. The slight grayish-white discoloration of the wall of the v. nas. sup. already mentioned had markedly increased. Over a space 1 PD in length the wall of the vein had been converted into an evenly broad, shining, white band ; at a point near the centre the red wall of the vein still shone through. Centrally from this spot, as far as the OD, the vein was very narrow, a mere thread, a sign therefore that the calibre had been decidedly diminished. Peripherally from the white spot on the contrary the vein was distended to twice or three times the normal, and was of a dark-red color. In tracing the ramification of the v. nasal. sup. toward the periphery, two more spots were found, showing similar changes, but not so extensive ; diminution of the calibre centrally from the diseased spots was not apparent. The most peripheral ramifications showed a perfectly cirsoid tortuosity, which could not be reproduced in the drawing, as besides the curves in the plane of the retina there were others rising above it, as shown by parallactic displacement. All the tortuous parts were filled beyond the normal. At the periphery, as the sketch indicates, there were some irregular, slightly brownish-red venous hemorrhages.

All the other veins, except the v. temp. super., showed similar changes.

The interpretation of the picture was not difficult. An infiltration of round-cells had developed, partly in the perivascular lymph spaces, partly in the wall of the vein itself, which could not be seen more beautifully. With the ophthalmoscope the progressive changes could be observed with almost microscopic accuracy. We saw distinctly how the shining white, band-like changes in the blood-vessels gradually developed from the small whitish spots; from the peripheral swelling, the tortuosity, and the gradually appearing hemorrhages, as well as from the central diminution of the blood-vessel, we could obtain a pretty accurate idea of the permeability of the affected part.

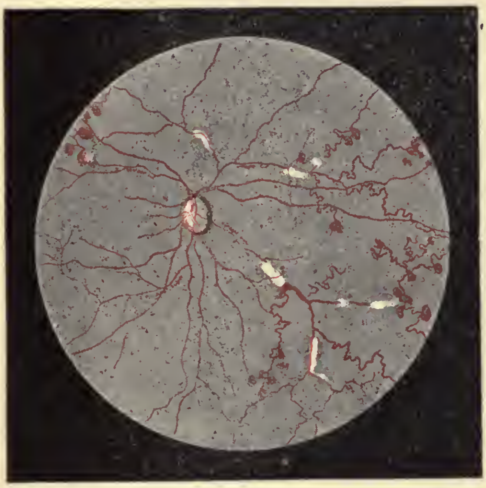

FIG. I.

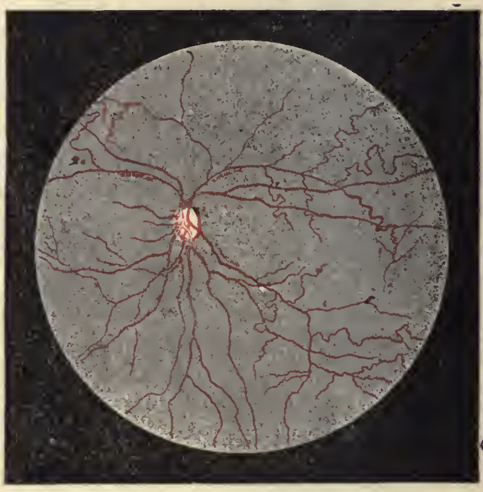

FIG. 2.

I have chosen the sketch made on February 28th for publication, because on that day the affection had reached its height.

The rapid further development of the disease, in spite of the energetic antisyphilitic treatment, might have caused us momentarily to become doubtful as to the correctness of the diagnosis. The father of the patient, however, who now appeared in answer to our wish, admitted that he had become infected after his marriage, and after his wife had already borne him four healthy children. When the dermic symptoms began to manifest themselves he underwent inunction and was cured. His wife thereupon had two abortions; then the son, the patient under our treatment, was born, and afterwards two more children. The latter are quite healthy. There could now be no more doubt about the correctness of the diagnosis. In spite of the rapid development of the affection, we therefore made a good prognosis, and were not deceived.

New hemorrhages and changes in the walls of the blood-vessels did not take place. As tumultuously as the disease had begun, just as rapidly the changes disappeared. Gradually the whitish discoloration became less and less marked, the red spots shining through became more and more numerous, and the filling of the blood-vessels centrally and peripherally from the affected parts more and more approached the normal. The hemorrhages were gradually absorbed, some after having undergone fatty degeneration. Three weeks later, on March 21st, V in the right eye had risen to $\frac{6}{18}$, on March 30th to $\frac{6}{12}$. In the left, normal sight had always been preserved. On April 8th, after 200 grms. of ungt. hyd. had been used, the patient was discharged from the hospital. R, V = $\frac{6}{9}$; L, = $\frac{6}{6}$.

Six months later, on October 13, 1888, the patient was seen again. V was the same. In the right eye there were still very thin remnants of hemorrhages, about $\frac{1}{4}$ PD in diameter, with delicate changes in the pigment at the macula, and a small whitish discoloration of the retina beside the v. temp. sup. for a distance of about $\frac{1}{3}$ PD; the calibre known was again normal. All the other hemorrhages had been completely absorbed. Here and there some patches of the pigment the size of a pinhead still remained at the periphery. The tortuosity of the peripheral veins, however, was the same as before, but the filling was not abnormal.

The condition of the left eye is shown in Fig. 2. Small pigment patches are the only evidences of the former hemorrhages. At the spot first infiltrated there is a delicate, grayish-white discoloration beside the once more normal v. nasal. sup. There are no other perivascular changes. As in the other eye the cirsoid tortuosity of the terminal veins has remained. But the calibre of the blood-vessels is normal with the exception of the endings of the v. nasal. infer., which shows besides greater tortuosity also greater filling with blood. Following this vein farther, three smaller veins are seen to join it ; from the manner of the junction it becomes apparent that the direction of the flow was formerly different. Now, however, the ending of the vein cannot be traced farther ; it disappears. Evidently the plaque had closed the calibre of the v. nasal. sup. at this point, and a collateral anastomosis with the v. nasal. inf. had been formed, an observation which has been made at various times with the ophthalmoscope.

On March 23, 1890, two years later, I examined the patient for the last time. R E, $V=\frac{6}{6}$; L, the same. In the right eye the hemorrhage at the macula had been completely absorbed. In the upright image about $\frac{1}{2}$ PD upward and inward from the macula a roundish spot the size of a pinhead is seen, of a brighter rosy-red color than the remainder of the retina, with a delicate darker edge, the only trace of the hemorrhage. Otherwise the condition is the same as on October 13, 1888. The veins in particular show the same cirsoid tortuosity at the periphery, their calibre being normal.

This latter condition also prevails in the left eye. The anastomosis has remained, and the three small veins discharge their blood into the v. nasal. infer. That part of the v. nasal super. which had been obstructed by the plaque had become so atrophic that it could not be found with the ophthalmoscope. The perivascular changes have completely disappeared.

In conclusion let me briefly sum up the peculiarities of the case. Congenital syphilis first manifested itself in a a strong, young blacksmith of eighteen in the form of perivasculitis of the retina of both eyes, attacking only the veins with the exception of the left v. tempor. super., leading to the partial occlusion of the calibre of the vessels, enormous dilatation, and very peculiar tortuosity of the terminal veins, and extensive hemorrhages, and quickly healing after inunction. The hemorrhages and perivascular patches are rapidly absorbed, only the curious tortuosity of the veins remains, but their calibre is normal. In the left eye an anastomosis develops between the vv. nasal. super. and infer., the result of occlusion of the blood-vessels by a pigmented plaque arising from a hemorrhage. Two years later the condition is practically the same.

From its first appearance to complete recovery the disease was observed with the ophthalmoscope.

Explanatory remarks as to the nature of the pathological changes are unnecessary, for since the publications of the articles of Michel[1] and Angelucci[2] on the spontaneous thrombosis of the central retinal vein peri- and endovasculitis have received general attention. As already mentioned and as shown by the case of phlebitis ophthalmica observed by Schmidt-Rimpler,[3] we had to deal with an infiltration of round-cells into the perivascular lymph spaces and into the walls themselves of the blood-vessels. Secondarily thrombosis and hemorrhages may result.

As early as 1878 Hutchinson[4] expressed the opinion that in a number of cases of hemorrhagic retinitis venous thrombosis might be the cause. Angelucci in many cases attributes the thrombosis to primary changes in the walls of the blood-vessels. In 1881 Schubert[5] publishes a case of hemorrhagic retinitis, which he also attributes to venous thrombosis.

Perivasculitis of the retinal arteries has been seen and described in a number of cases. Fürstner[6] has published such a case, which Becker observed with the ophthalmoscope, in which there was arteriitis obliterans of the vertebral, basilar, and ophthalmic arteries with slight implication of the ciliary and retinal arteries. The existence of syphilis could not be established. In the case described by Hirschberg,[7] however, syphilis was the cause, and Haab[8] also observed and published a case of syphilitic arteriitis.

I am greatly indebted to Professor Pagenstecher for his suggestions and kind assistance.

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[1] 1878. Die spontane Thrombose der Vena centr. des Opticus, *Graefe's Archiv*, vol. xxiv., 2, p. 37.

[2] 1878. Thrombose der Vena centr. retinae, *Klin. Monatsbl. f. Augenheilk.*, p. 443.

[3] 1877. Phlebitis ophthalmica, *Graefe's Archiv*, p. 229.

[4] 1878. *Med. Times and Gazette*, vol. i., p. 104.

[5] 1881. Zur Casuistik der Retinitis syphil., *Centralbl. f. prakt. Augenheilk.*

[6] 1883. *Deutsche Zeitschr. f. klin. Med.*, vol. xxx., p. 534.

[7] 1883. *Centralbl. f. prakt. Augenheilk.*

[8] 1886. *Schweizer Correspondenzbl.*, No. 6, p. 152.

A CASE OF UNIOCULAR ZONULAR CATARACT.

BY F. E. D'OENCH, M.D.

About five years ago, Louis S., æt. eight, was brought to the Dispensary of the New York Ophthalmic and Aural Institute on account of defective vision of his left eye, which only lately had been discovered. No complaint was made in regard to the right, except that the boy in reading was obliged to hold the book quite near the eye.

The cause of the poor sight of the left eye was found to be due to a zonular cataract, the diameter of the opaque zone being about 3‴, and showing an irregular whitish thickening at the anterior pole. The remainder of the lens was clear, $V = \frac{1}{\infty}$. The other eye showed no changes whatever, the lens in particular being perfectly clear. The child had never had convulsions, and his teeth were good, without any notches.

An iridectomy downward and inward was performed by Dr. Knapp, after which vision rose to $\frac{8}{200}$.

The boy was then lost sight of, and I did not see him again until five years later, when he came back to have his right eye fitted with glasses. There has apparently been no change in the zonular cataract, but vision has diminished, being only $\frac{2}{200}$, which with $-11\ D^c$ ax 65°n could be raised to $\frac{4}{200}$. The lens is of normal size, and its edge can be distinctly seen at the periphery of the coloboma.

The vision of the right eye is $\frac{15}{200}$, which with $-4\ D \bigcirc -4\ D^c$ ax 70°n can be raised to $\frac{20}{20}+$; with $-4\ D^c$ ax 70°n he can read J 1 at about 9″, and these two glasses were prescribed for him, the left receiving only a plane glass.

The absence of convulsions, and of any change in the cataract for five years, make it very probable that the condition is a congenital one.

A BLACK SPOT AT THE MACULA LUTEA IN A CASE OF EMBOLISM OF THE ARTERIA CENTRALIS RETINÆ.

By Dr. T. INOUYE, Tokio, Japan.

Myjabe, a girl, æt. seven, consulted me October 19, 1890, with the following history.

Three years previously she had suffered from blennorrhœa which had caused blindness in the left eye. At the same time she became emaciated, and exhibited a morbid appetite, devouring greedily mud, linen, etc.

October 17th, on rising in the morning she found that she was completely blind, the right eye also having become affected. The patient felt well in other respects, and there was no heart lesion. In the right eye all perception of light was lost, but the exterior of the eye appeared normal. With the ophthalmoscope it was at once noticed that the usual transparence of the retina was wanting. In consequence of this the pale optic disc was not sharply outlined. The arteries and veins were not clearly distinguishable, and it was difficult or almost impossible to say which were the arteries and which the veins. All the veins were narrow and thread-like, showing in the middle a barely perceptible light-reflex. The arterial current was interrupted and slow. Between the separate blood-cylinders the vessel tube appeared as a bright thread-like line. At the macula lutea a small coal-black spot was seen in place of the usual cherry-red one, so well known in embolism.

This color interested me, since in the Japanese I had never before found the spot black, but always red. The Japanese are strongly pigmented, and it is probable that the black choroid shone through the thin fovea centralis, although the child was not unusually dark.

I have found no case similar to this in literature, and I am curious to know whether in cases of embolism in the negro the macula lutea appears black or has its usual cherry color.

A CASE OF LENTI-CONUS POSTERIOR, WITH REMARKS.

By JOHN E. WEEKS, M.D.,

SURGEON TO THE NEW YORK EYE AND EAR INFIRMARY.

M. L., age seven years. Has had convergent strabismus of the left eye since she was an infant. The patient was brought to my clinic at the New York Eye and Ear Infirmary on February 28, 1891, for the purpose of having the squint corrected. On examination the following conditions were found :

Right eye.—From the line between the greater and minor zones of the iris two small filaments project, extending into the pupil. Remnants of the fœtal pupillary membrane. In other respects the eye is perfectly normal. $V = \frac{20}{20}$.

Left eye.—Size normal ; pupil central, of normal size, reacts well to light. On looking into the eye with the ophthalmoscope, using the mirror only, a bright circular patch, apparently about 4 *mm* in diameter is seen, located between the iris and the fundus in the antero-posterior axis of the globe. About the margins of this disc light and dark crescentric figures play on changes in the direction of the light. When viewing the interior at about twelve inches from the eye, images of the vessels of the fundus are seen which on movements of the observer's head appear to go with or against it according as the vessels are seen through the periphery or through the centre of the lens. Hence at the centre of the lens the eye is myopic, while at the periphery of the lens it is hypermetropic.

If while holding the mirror in one position the cornea is made to move in various directions, the disc is seen to move, and, when compared to the position of the corneal reflex (which is always referred, by the observer, to the centre of curvature of that part of the surface of the cornea from which it is seen—in normal corneæ to a point approximately 7.8 *mm* from the surface), the disc moves with the cornea (apparently against the reflex), the periphery of the disc describing a larger arc than the centre, in fact the centre or apex of this body corresponds almost exactly with the centre of curvature of the cornea. It follows that this mass projects beyond the normal curvature of the lens, and since the posterior surface of the lens is normally about 7.2 *mm* from the surface of the cornea, 0.6 *mm* less than the length of the radius of the cornea, it has an antero-posterior diameter of about 0.5 *mm.* Its base is probably about 2.5 *mm* in diameter. The condition is known as lenti-conus or cristallo-conus posterior. At the apex of the cone there is a small opacity which may be, and from its flattened form most probably is, a remnant of fœtal blood-vessels. In all other respects the interior of the eye is normal. Vision $= \frac{1}{200}$; not improved with glasses. At the periphery of the lens the refraction is hyperopic 3.5 D, at the centre myopic about 12 D. The strabismus measures twenty-six degrees. The excursions of the eye are normal in extent.

Cases of lenti-conus are very rare, four only having been reported. The first is by Webster (*Arch. of Ophthal.*, 1875), and is one of binocular lenti-conus anterior. It occurred in a male of twenty-four years. The vision in either eye was $\frac{15}{100}$ increased to $\frac{20}{100}$ with glasses. Illumination with the ophthalmoscopic mirror gave the appearance of kerato-conus. At a distance of twelve inches two images of retinal vessels could be seen, one in the centre of the pupillary reflex and one at the periphery. On movement of the observer's head from side to side the images were seen to move in opposite directions. Examination by oblique illumination revealed a conical protrusion of the centre of the anterior part of each lens into the anterior chamber. The whole surface of the posterior capsule was dotted over with minute opacities, and there was small posterior polar cataract.

The second case is reported by Van der Laan and Placido. (*Period. de Ophthal. Lissboā*, 1881).

As I have not succeeded in obtaining the original article, I cannot do better than to refer to the abstracts contained in these ARCHIVES, vol. x., p. 497, and in *Nagel's Jahresbericht*, 1880, p. 369.

The case was one of lenti-conus anterior occurring in a man of 23 years. Whether one or both eyes were affected is not stated. The myopia at the apex of the cone was of a high degree, at the periphery of the lens the refraction was hypermetropic three (3) dioptries; a history of gradual development during a period of eight years was given.

The first case of lenti-conus posterior was reported by F. Meyer (*Hirschberg's Centralbl f. p. Augenhl.* Bd. xii., p. 41), and occurred in a boy of ten years. For three or four years the father had noticed that the right eye turned outward. When seen there was moderate divergent strabismus of the right eye. Vision $= \frac{13}{200}$, not much improved with glasses. Left eye, vision $= \frac{20}{20}$; Em. On looking into the right eye with the plane mirror of the ophthalmoscope, a circular spot was seen in the centre of the pupil, circumscribed by light and dark rings, which would change their position on a change in the direction of the light. At the apex of the cone there was a sharply defined opacity, and a number of small opacities were present on the posterior capsule near the posterior pole. These spots appeared white by oblique illumination. At the periphery of the lens the refraction was emmetropic, at the centre myopic 13 D. The eye was normal in size.

The second case of lenti-conus posterior is reported by Dr. H. Knapp (these ARCHIVES, vol. xviii., p. 453), and occurred in a girl of eight years. The left eye was normal in every respect. There was binocular fixation when regarding objects attentively, but soon the right eye would swing 3 or 4 *mm* outward. Vision $= \frac{12}{200}$ with — 12 D $\frac{20}{200}$. Fundus normal. " With a plane mirror a sharply defined red disc, of an apparent diameter of 4 *mm*, is seen in the centre of the red pupil, surrounded by dark shadows. It resembles an oil globule on water." In relation to the corneal reflex, the disc moves with the cornea on movements of the eye. A double set of vessels may be seen moving in opposite directions on movements of the observer's head. There is no opacity in lens or capsule. The eye is normal in size.

The manner in which lenti-conus is produced is as yet obscure. Prof. O. Becker (" Anatomie d. gesunden u. kranken Linse," 1883, p. 126) has made a very interesting report of the examination of specimens of lenti-conus posterior from the eyes of a rabbit sent to him by Schöler and Uhthoff, of Berlin. Although the sections obtained were not as perfect as was desired, Becker was able to determine that the concentric lens fibres, products of the third period of lens development, did not come together at the posterior pole, but that they butted on to, or formed around, the elongated nuclear fibres of the second period of lens development, these latter extending beyond the concentric laminæ and forming the cone.

It may readily be conceived that traction on the anterior or posterior parts of the lens in early fœtal life might produce this condition. In lenti-conus anterior it would only be necessary to infer that the lens had become unusually adherent to the posterior surface of the cornea in its early embryonic stage, and that in the formation of the anterior chamber some traction had been exerted. In the cases observed no mention is made of any irregularities in cornea or lens that could be construed as facts in favor of this supposition. In lenti-conus posterior the conditions are somewhat different. In my own case and in the case described by Meyer, there was evidence of the unusual persistence of fœtal tissue in the presence of opacities on the posterior lens capsule. Opacities in each case were situated at the apex of the cone, and may have been the site of attachment of fœtal vessels which, by producing traction on the forming nuclear fibres of the lens, had caused them to become unusually elongated. Lenti-conus is probably always congenital.

ON THE USE OF PLATINUM INSTRUMENTS IN OPHTHALMIC SURGERY.

By Dr. E. GRUENING, New York.

IN bacteriological technique metallic instruments, notably platinum needles, are heated to a glow and thus sterilized. This ideal depuration cannot be practised in general surgery, inasmuch as both the bulk and the material of the instruments forbid a similar treatment. In ophthalmic surgery, however, where the instruments are comparatively small, the intractable steel may be replaced by the more easily managed platinum.

Messrs. Tiemann & Co., made for me a set of platinum eye-instruments which have been in use almost one year. The set contains a cystitome, an iris repositor, a wire loop, a spoon, several straight and curved iris forceps, a fixation forceps, and a speculum. The working parts of these instruments consist of an alloy of platinum and iridium. The platinum, thus alloyed and compressed by hammering, becomes very firm, and when polished is not blackened by the flame of a spirit lamp.

Incomplete as the set is (it contains neither knives nor scissors), it stood me in good stead, and to-day I should not like to perform an operation for the extraction of cataract without the aid of these instruments.

The time required for the sterilization of a platinum instrument by imparting to it a white heat is but a small fraction of a minute, so that every instrument required for an operation can be prepared in a direct, simple, and thorough manner by the surgeon. This feature, which renders the operator independent of an assistant in the sterilization of the instruments, is of special importance.

When we consider that the structures of the eye, notably the cornea and the vitreous body, furnish a favorable nutrient soil for the development of micro-organisms, and that the introduction of these into the eye is in many instances tantamount to its destruction, it becomes evident that the use of platinum instruments is not an unnecessary refinement.

It may be argued that suppuration after operations upon the eye is rare at the present time, and that the statistical records of skilled operators show but a small percentage of loss from either primary or secondary infection.

All this is conceded, but I hold that our statistics should be improved still further, so that our future records might show no loss from suppuration and a much less frequent occurrence of non-suppurative inflammations, such as iritis, irido-cyclitis, and capsulitis.

I believe that the more general use of platinum instruments may aid in insuring such grateful results—" a consummation devoutly to be wished."

PRISM MEASURE OR LENS CENTERING INSTRUMENT.[1]

By A. L. SMITH, Pres. Geneva Optical Company.

WE illustrate herewith a new device for the use of opticians and oculists for the purpose of centering lenses and also for measuring the degree of a prism, or prisms in lenses combined with spherical or cylindrical sur-

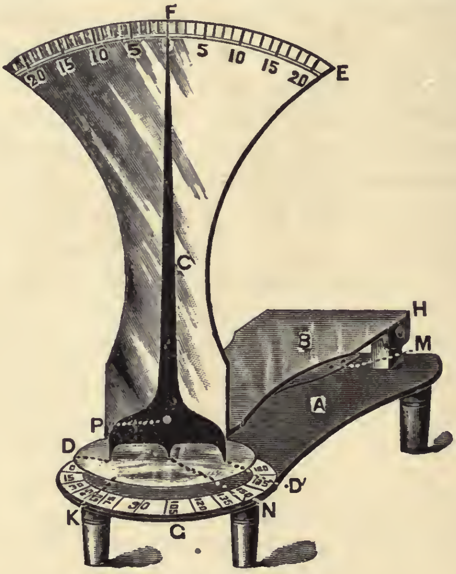

FIG. I.

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[1] Mr. Smith, who designed the instrument himself, was kind enough to send me a sample of it with the above communication. On examining and testing it, I find that it answers its purpose in every respect. Like the lens measure, it will prove a valuable aid in the daily work of the oculist. While thanking and congratulating the Gen. Opt. Co., I cannot refrain from expressing my pleasure at seeing that the spirit of enterprise and ingenuity, for which American industry is so famous, has become fully alive also in the department of opthalmological optics. H. KNAPP.

faces. The instrument is so simple in its action that a description is hardly necessary. The illustrations will make plain to any oculist or optician the entire practicability and range of its work. It will give instantly the amount of prism in any lens, and the value of such an instrument will be appreciated by the trade and profession, as it is one of the most difficult and annoying parts of the business to analyze a lens and give the amount of prism, especially when combined with convex or concave surfaces. Its action will be readily understood from the following description.

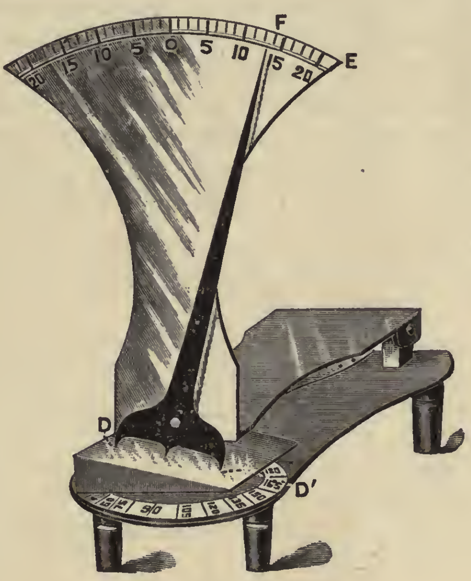

FIG. 2.

The first illustration gives the instrument as used for centering lenses. The instrument consists of a bed plate *A*, upon the front of which is affixed a degree circle *G*, and hinged to *A* at *H* is the upper plate *B* thrown up by a spring *M* not shown plainly because it is under *B*. The upright face plate *C* stands at right angles to *B*. On the top of *C* is the degree circle *E*. The index finger *F* with lower part *D D′* is made of steel and pivoted at *P* to swing easily over any portion of the dial plate. In using, the lens *K* is placed upon the lower points that cannot be shown, as they are covered by the lens. The upper plate *B* is pressed down until the points *D D′* touch the lens, and if the lens is of the same thickness at *D* as at *D′* the index finger *F* will point to *O* on the scale *E* and the point *N* will be exactly over the centre of the lens.

The second illustration shows the same instrument measuring a prism, and the position of the index finger *F* will be governed by the difference of the thickness of the lens at the points *D D′* and the degree of the prism will be indicated on scale *E* by the pointer *F*. The operation is quick and exact, and the value of the instrument at the present time, when so much attention is being given to the correctly placing the centre of the lens or adding prism to correct for muscular defect, makes this an invaluable instrument for the oculist or optician. Handsomely made in nickel with steel pointer. It is manufactured and patented by the Geneva Optical Co., 23 Washington St., Chicago, and the price is $5.

SPONTANEOUS CURE OF PROLAPSE OF IRIS AFTER CATARACT EXTRACTION.

By Dr. E. LOPEZ, of Havana, Cuba.

I USUALLY operate for cataract by simple extraction with superior keratotomy. One of the most disagreeable complications of this method is the occurrence of prolapse of iris. The usual treatment of these prolapses with myotics, has, as a rule, no effect. In these cases the excision of the prolapsed portion seems to be rational. Nevertheless there are a few cases, similar to those which I am about to describe, in which it would seem proper to temporize a little with this complication, as the prolapse may disappear without surgical interference.

1st Case.—Woman; fifty years of age; simple extraction (R. E.); no accident; eserine (1%); compressive bandage over both eyes.

5th day.—Good condition; apparently a firm scar; pupil large, somewhat elongated upwards, pyriform.

7th day.—Conjunctival injection; prolapse of iris at the centre of the section; the prolapse is small; eserine.

12th day.—The iris has re-entered the anterior chamber, remaining attached to the scar at its base, leaving a slightly oval pupil.

2d Case.—Woman; fifty-eight years of age; simple extraction (R. E.); no accident; eserine (1%); round pupil; both eyes bandaged.

4th day.—Small prolapse of iris; pain in the right temple; atropine ($\frac{0.02}{10.00}$).

15th day.—The prolapse has disappeared, leaving only the base of the iris attached to the internal surface of the scar. The pupil is slightly distorted and partly obstructed by secondary opacities.

In these cases the spontaneous cure of these small prolapses is proven. This can in nowise be attributed to the treatment, for although in the first case myotics were employed, in the second mydriatics were made necessary by the incipient iritis.

SUPPLEMENT TO DR. GEO. LINDSAY JOHNSON'S PAPER: A NEW TREATMENT OF CHRONIC TRACHOMA.

PUBLISHED IN THE PREVIOUS VOLUME OF THESE ARCHIVES (XIX., P. 264).

(*With plate ii. of vol. xix.*)

DR. JOHNSON illustrated his article by two chromolithographs on Tab. II., which ought to have been issued with his paper. They are appended to the present number, and we beg our readers to make a note of it in their copies. The explanation of the plates is as follows:

The two drawings present the right and left upper eyelid of a ten-year-old girl. Both lids were in the same condition, affected in the same manner with follicular trachoma. The operation described in the paper was performed on the left eye (Fig. 2). Six weeks later both eyes were drawn (Figs. 1 and 2). The white horizontal stripes (Fig. 2) represent tracts of cicatricial tissue caused by the wounds from the action of the sillonneur. The immediate union of these wounds had been prevented by the electrolysic destruction brought about by the platina plates.[1]

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[1] The editor takes pleasure in stating that last summer he had the privilege of seeing Dr. Johnson operate on two cases, and to witness the highly satisfactory recovery of others which had been operated on by the doctor, according to his method.

H. K.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FIRST HALF OF THE YEAR 1890.

By H. MAGNUS, Breslau; C. HORSTMANN, and
P. SILEX, Berlin.

WITH THE CO-OPERATION OF

S. M. Burnett, Washington; Dantone, Rome; Hirschmann, Charkow;
E. Marckwort, Antwerp; P. v. Mittelstädt, Metz; L. Werner,
Dublin; C. H. A. Westhoff, Amsterdam; Schiötz and
Ole Bull, Christiania, etc.

(Continued and concluded from p. 159 of this volume.)

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

103. Burchardt, M. Gonorrhoic ophthalmia, especially its treatment. *Deutsche militärärztl. Zeitschr.*, vol. xix., 8, p. 592.

104. Deutschmann, R. Arthritis blennorrhoica. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 1, p. 109.

105. Franke, E. The transplantation of skin and mucous membrane. *Centralbl. f. prakt. Augenhk.*, 1890, p. 161.

106. Germann, Th. The etiology of trachoma. *St. Petersburger med. Wochenschr.*, 1890, No. 29.

107. Kipp, C. J. Further observations on malarial keratitis. *Trans. Amer. Ophth. Soc.*, 1889.

108. Krause, P. The history of trachoma since the wars of Napoleon to the end of the year 1889. *Inaug. Diss.*, Berlin, 1890.

109. Lippincott, J. A. Irrigation of the anterior chamber—a new syringe for effecting it. *Trans. Amer. Ophth. Soc.*, 1889.

110. Noiszewski, K. Plastic conjunctivitis—a radical method of curing chronic trachoma. *Centralbl. f. prakt. Augenheilk.*, 1890, p. 36, No. 234 ; *Wratsch*, 1890, No. 29.

111. Rosmini. Sull' uso del sublimato corrosivo nella terapia oculare e specialemente nella cura dell' oftalmia granulo-tracomatosa. *Boll. d'ocul.*, vol. xii., Nos. 7, 9, and 11.

112. Schmidt-Rimpler, H. Remarks on the etiology and treatment of blennorrhœa neonatorum. *Deutsche med. Wochenschr.*, 1890, No. 31.

113. Shongolowicz, D. The question of the micro-organism of trachoma. *St. Petersburger med. Wochenschr.*, 1890, Nos. 28 to 30.

114. Valenta, A. Contribution to the prevention of ophthalmo-blennorrhœa neonatorum. *Wiener klin. Wochenshr.*, 1890, No. 35.

In gonorrhoic conjunctivitis Burchardt (103) thoroughly washes out the conjunctival sac with a salicylic solution (1.5:1000), and then with a solution of nitrate of silver (1:600). He also instils a weak solution of nitrate of silver every two hours.

Deutschmann (104) observed acute inflammation of the knee-joint in a child, three weeks after a genuine blennorrhoic conjunctivitis. Both the conjunctival discharge and the liquid obtained by puncture from the knee-joint contained typical diplococci. Microscopic examination of a piece of conjunctiva showed gonococci upon and between the epithelial cells, and in the subepithelial tissue enclosed in pus-cells, close to the walls of the delicate loops of blood-vessels extending to this point. The cocci probably enter the blood- and lymph-vessels at this point and infect those tissues which react especially to their presence.

Franke (105) cured a case of trichiasis by transplantation of a piece of skin from the upper arm.

Germann (106) observed in three cases the development of trachoma after some black earth had entered the conjunctival sac. He therefore thinks that dirt and dust contain the micro-organisms producing trachoma. The intensity of the inflammation and the length of the stage of incubation seem to depend upon the quantity of germs introduced.

Since Kipp (107) first reported on malarial keratitis in 1880 he has studied 120 additional cases. He finds that fully 90% were affected with malarial fever. He has, however, seen the peculiar dendritic ulceration in persons who were perfectly healthy. He has tried scraping the ulcers, and scraping with application of nitrate of silver, in some cases with benefit. He attaches most importance to general treatment of the malarial poisoning. BURNETT.

LIPPINCOTT (109) has used irrigation of the anterior chamber in fifteen cases of cataract-extraction and had satisfactory results. His instrument consists essentially of a rubber tube with a fine gold nozzle attached to it, with a holder and a sort of valve, which regulates the flow of the water from a reservoir placed above, and in which the other end of the tube is placed. He uses any aseptic fluid, a strong antiseptic not being desirable.

BURNETT.

In trachoma NOISZEWSKI (110) excised the retrotarsal fold and the affected part of the conjunctiva, and implanted a piece of mucous membrane taken from the lip. He recommends this method in all cases of old trachoma; it should not be resorted to when the ordinary methods of treatment are sufficient.

ROSMINI (111) is very enthusiastic over the action of sublimate in follicular and trachomatous conjunctivitis, and applies it in a solution of 1:1000 (sometimes also weaker) with an irrigator to the affected mucous membrane for thirty to sixty seconds and longer. He lays great stress upon this method of application.

DANTONE.

SCHMIDT-RIMPLER (112) calls attention to the fact that frequently cases of blennorrhœa neonatorum are observed in which no gonococci are found in the discharge, and the course is, nevertheless, a severe one. He therefore recommends the prophylactic treatment of the eyes of the new-born whenever there is fluor, even if not of a gonorrhoic nature. Simple ablution with water is not sufficient; antiseptic instillation into the eyes affords greater security. To avoid irritation he uses the officinal aqua chlori, the disinfecting action of which is very great. It is well borne by the conjunctiva.

SHONGOLOWICZ (113) examined the contents of the follicles of 26 patients with well-developed trachoma, and also in 12 of them the retrotarsal folds. He found in all cases in the contents of the trachoma follicle among the micro-organisms present particularly short rods, remarkable on account of their small size and the difficulty of staining them with aniline colors; all parts of the rods were not stained with equal intensity and the refraction in the different parts varied; the same short rods were found in sections of the fornix of trachomatous conjunctiva, while other micro-organisms were absent. Sometimes an affection similar to trachoma could be produced upon the conjunctiva of cats and rabbits by inoculation with pure cultures.

VALENTA (114) uses a weak solution of permanganate of potassium.

115. BETTREMIEUX. Considérations sur le traitement de la cornée à hypopyon. *Soc. franç. d'opht.*, May 6, 1890.

116. BRAUNSCHWEIG. A contribution to the knowledge of pyoktanin. *Fortschritte d. Med.*, 1890, No. 11.

117. CZERMAK, W. On vesicular cavities and their relation to corneal fistula. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 2, p. 163.

118. GALEZOWSKI. De l'epithélioma cornéen et son traitement. *Soc. d'opht. de Paris*, Jan. 7, 1890.

119. GALEZOWSKI. Epithélioma de la cornée. *Progr. méd.*, 1890, No. 4.

120. HIRSCHBERGER. On corneal affections in influenza. *Münchener med. Wochenschr.*, 1890, No. 4.

121. LANG and WOOD. An examination of the patellar tendon-reflex in 62 cases of interstitial keratitis. *Royal London Ophth. Hosp. Rep.*, vol. xii., p. 312.

122. LAWFORD, J. B. Remarks on keratitis punctata or descemetitis. *Royal London Ophth. Hosp. Rep.*, vol. xii., p. 298.

123. PERA. Contributo alla cura delle ulceri della cornea. *Boll. d'ocul.*, vol. xii., 10–11.

124. PFISTER, J. One hundred and thirty cases of keratitis interstitialis diffusa, including five cases of keratitis interstitialis annularis according to Vossius. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 114.

125. SCHEFFELS, O. On the treatment of corneal ulcers with pyoktanin. *Berliner klin. Wochenschr.*, 1890, No. 28.

126. STILLING, J. Aniline dyes as antiseptics and their application in practice. Strassburg, 1890.

127. TAYLOR, C. B. On the importance of securing coaptation of the lids in certain diseases of the eye. *The Lancet*, Feb., 1890, p. 293.

128. WERJINSKI, A. Diffuse parenchymatous inflammation of the cornea. *Medic. Obosernje*, 1890, vol xxviii., No. 9.

According to BETTREMIEUX (115) it frequently happens in corneal ulcer with hypopyon that the suppuration spreads in spite of antiseptics and Saemisch's incision. In such cases an iridectomy proves useful. According to Dufour this has long been known. MARCKWORT.

BRAUNSCHWEIG (116) from his experience warns against the use of pyoktanin, as in several cases it did direct harm. SCHEFFELS (125) also had only negative results in corneal ulcers. He considers the treatment with pyoktanin harmful, because time is lost which might have been better employed by dividing or cauterizing the ulcer.

CZERMAK (117) describes four cases in which vesicular cavities had developed in corneal scars. In all of them they were the result of perforating ulcers. The condition of the outer surface was favorable to bursting, and a canal discharging the aqueous externally was found. For details see the original.

GALEZOWSKI (118) discusses epithelioma of the cornea, and on the basis of a reported case recommends the application of the galvano-cautery to the wound and its surroundings after the removal of the tumor. Dubois de Lavigerie used lapis infernalis instead of the galvano-cautery, and had no relapse. Chibret prefers the thermo-cautery for this purpose on account of its more energetic action. MARCKWORT.

In the 62 cases examined by LANG and WOOD (121) hereditary syphilis existed in 69%, and struma in about 11%. In 30% of the cases the patellar reflex was subnormal, and entirely absent in 10%. WERNER.

LAWFORD (122) believes that the cells in the deposits of keratitis punctata are migratory cells, and that the underlying cells of Descemet's membrane become indistinct and swollen.

WERNER.

PERA (123) reports a case of corneal ulcers of both eyes due to trachoma, in which even the weakest solutions of sublimate (1 : 5000 and 1 : 7000) were not tolerated, and only caused pain and increased irritation. He therefore concludes that sublimate should only be used in extensive ulcers at an advanced stage of repair. DANTONE.

PFISTER (124) found that among 130 cases of keratitis punctata the affection occurred in both eyes in 69.2%. Hereditary syphilis undoubtedly existed in 40%, probably in 23.8%, acquired syphilis in 2.3%, probably acquired syphilis in 1.5%. Hutchinson teeth were observed in 23.8%, choroiditis disseminata was found in 6.9%, serous iritis in 13%, and plastic iritis in 6.9%.

STILLING (126) recommends methyl-violet in corneal ulcers and hypopyon-keratitis, as it is a powerful antiseptic. He also advises its application in keratitis parenchymatosa, serous iritis, old cases of disseminate choroiditis, and in syphilitic ophthalmia. He used the remedy in solution, and in form of a powder, salve, and pencil.

TAYLOR (127) in a case of corneal ulceration from Basedow's disease, united the lids, leaving a central slit-like portion free.

WERNER.

129. MÜLLER. Case of tuberculosis of the sclera. *Wiener med. Blätter*, 1890, No. 12.

130. ROLLAND. Extraction d'un tumeur mélanotique intra-oculaire par une incision de la sclérotique. *Rec. d'opht.*, 1890, p. 25.

In MÜLLER'S (129) case there was a cystic tumor upon the sclera, which had been mistaken for a cysticercus and removed. It proved however to be a conglomeration of miliary tubercles.

ROLLAND (130) found in a man aged 32, upon the upper inner part of the eye, 6 *mm* from the corneal margin, a tumor the size of a bean upon the outer surface of the sclera, and at the same spot within the eye another the size of a fly's head. Vision was normal. The two tumors were removed together with the small piece of sclera separating them. The opening in the sclera was closed with a catgut suture, and that in the conjunctiva with three silk sutures. The clinical diagnosis of melanosarcoma was verified by the microscopical examination. Seven months after the operation the family physician reported to Rolland that the patient "enjoyed perfect vision and health."

MARCKWORT.

Sections XII.—XXI. Reviewed by DR. P. SILEX.

XII.—IRIS.

131. BURCHARD. Presentation of a case of paradoxical pupillary reaction. *Berliner klin. Wochenschr.*, 1890, p. 40.

132. GUTTMANN, G. A case of tuberculosis of the eye. *Berliner med. Gesellsch.*, March 26, 1890. *Deutsche med. Ztg.*, 1890, No. 25.

133. LANG, W. The division of anterior synechiæ. *Royal London Ophth. Hosp. Rep.*, vol. xii., p. 356.

134. Panas. La tuberculose de l'œil. *Progr. méd.*, 1889, No. 46, p. 465. (Recommends the internal exhibition of iodoform [0. 4 daily] in tuberculous iritis.)

135. Terson (de Toulouse). Tuberculose oculaire, excision d'un tubercule de l'iris suivie de succès. *Arch. d'opht.*, vol. x., 1, p. 7.

136. Wagenmann, A. On pseudo-tuberculous inflammations of the conjunctiva and iris from caterpillar hairs. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 1, p. 126.

137. Williams, H. W. Multiple cysts of the iris in both eyes. *Trans. Amer. Ophth. Soc.*, 1889.

On Burchard's (131) patient iridectomy had been performed on account of total adhesion. When light was thrown into the eye the pupil dilated, and contracted when it was shaded. Burchard explains it by saying that the sphincter, which had been completely divided below, contracted upon illumination ; but, being firmly adherent above, it drew the three edges apart, thereby dilating the pupil.

In Guttmann's (132) case, a child six months old with hereditary syphilis, there was a large tuberculous tumor upon the iris of the right eye, and near it very small miliary nodules. In the left eye there was tuberculosis of the choroid.

Lang (133) makes a corneal puncture with a Knapp discission knife, which is withdrawn, and the synechiæ are then divided with a similar but blunt-pointed knife passed through the puncture.

Werner.

Terson (135) observed in an otherwise healthy one-year-old child the development of an iris-tubercle, which first appeared as a yellowish-white nodule the size of a pinhead at the periphery of the iris, with marked injection of the neighboring sclera, and the appearances of serous iritis. Four months later, when the tumor had grown into the anterior chamber, the author made a large peripheric flap-section with a Graefe knife, drew the nodule out, and cut it off, a little vitreous escaping. The tumor had probably united with the ciliary body, and the zonula had been torn and the lens dislocated. The tumor had the shape and size of a large pea. Healing progressed slowly but favorably. $V = \frac{1}{8}$; ophthalmoscopic condition not noted. Inoculation with the tuberculous mass and microscopic examination confirmed the diagnosis. The author thinks that as soon as the tumor appears in the anterior chamber it should be excised, except when the tumor springs from the periphery, when it is advisable to wait awhile, in order to avoid the danger of breaking up the tumor, which here is only accessible with difficulty, and thereby producing new sources of infection. He is opposed to enucleation, as it does not prevent general infection with certainty.

v. MITTELSTÄDT.

WAGENMANN (136) adds to the number of these cases as reported by Pagenstecher and Weiss.

WILLIAMS' (137) case was a girl of nine years, in whom there was a large single cyst and two smaller ones in the right, and two somewhat convoluted cysts extending into the pupillary space above and below in the left eye. Very little vision, and a good view of the fundus could not be obtained. Patient seen but once.

BURNETT.

XIII.—CHOROID.

138. DUCAMP. Un cas de sarcome ossifiant de la choroide. *Montpellier méd.*, 1889, p. 533.

139. EWING, E. Metastatic carcinoma of the choroid, the ciliary body, and the iris. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 1, p. 121.

140. NOYES, H. D. Enucleation during panophthalmitis suppurativa—what is the danger to life and how to obviate it? *Trans, Amer. Ophth. Soc.*, 1889.

NOYES (140) reports a case of enucleation after panophthalmitis had supervened and where there were symptoms pointing to serious brain implication. He made numerous and deep incisions into the orbital tissues, letting out pus and blood; antiseptic injections and dressings were used and the man recovered.

BURNETT.

XIV.—GLAUCOMA.

141. BULL, C. S. An analysis of 90 cases of simple glaucoma with special reference to the effects of iridectomy on the acuteness of vision and the visual field. *Trans. Amer. Ophth. Soc.*, 1889.

142. FALCHI, FR. On not congenital hydrophthalmus. Beiräge zur pathol. Anat. u. zur allgem. Pathol., vol. vii.

143 GRUENING, E. Iridectomy in glaucoma. *Trans. Amer. Ophth. Soc.*, 1889.

144. Logetschnikow, S. N. On some rare peculiarities of glaucoma. *Wjestnik Ophth.*, 1890, No. 3.

145. Nathanson, A. On glaucoma in aphakial eyes. (From the St. Petersburg Ophthalmic Hospital.) *Wjestnik Ophth.*, Jan., Feb., March, April, 1890.

It would not be possible to do justice to this (141) carefully prepared and succinctly tabulated paper in a mere abstract. It should be read in full to understand its value in regard to the very important question of the advisability of iridectomy in simple glaucoma; and though the matter is left but little in advance of where the author found it, yet this little may be of great value in deciding upon some single indication as to whether or not an operation should be done in some specific case. His cases seem to lend us somewhat more hope from the operation. Burnett.

Gruening (143) divides his cases for study into (1) acute inflammatory glaucoma; (2) chronic inflammatory glaucoma without visible degeneration of the iris; (3) the same with degenerative changes; (4) simple glaucoma; (5) intermittent glaucoma. In the first form irridectomy was highly satisfactory; in the second the results are also satisfactory; in the third neither iridectomy nor sclerotomy do good—posterior sclerotomy may accomplish something; in the fourth class, simple glaucoma, he thinks iridectomy should always be done. He does not believe the operation injurious, and it often stops the progress of the disease. In the fifth class, intermittent glaucoma, reduction of vision often results from iridectomy. Burnett.

Logetschnikow (144) reports a case of acute glaucoma with a preponderance of the symptoms of irido-cyclitis (glaucoma iridis, glaucoma anterior), which returned in spite of sclerotomy, disappeared after a subsequent iridectomy, and notwithstanding its long duration and marked reduction of sight (to $\frac{1}{14}$) had not led after three years to an excavation of the papilla; a case of glaucoma with distinct swelling of the papilla, which did not disappear after an iridectomy,—the latter, however, reduced the tension which had not been greatly increased; a case of acute glaucoma in both eyes with marked papillitis, which, twelve days after iridectomy and double seclerotomy, respectively, on the other eye, was followed by deep glaucomatous excavations.

Hirschmann.

Nathanson (145) has carefully analyzed twenty-eight cases of glaucoma in aphakial eyes thus far reported, and nine observed at the St. Petersburg Ophthalmic hospital. He divides them into two groups. In the first he places all cases in which the symptoms of glaucoma appear immediately or soon (a few weeks or months) after the operation, therefore depend upon the latter, and must be considered secondary glaucomas. In the second he places all cases, in which years have elapsed before the appearance of the glaucomatous symptoms. The cause of some of them may be found in the changes caused by the operation, which only an anatomical examination could reveal ; they belong therefore among the secondary glaucomas. In many cases the idiopathic character of the disease cannot be questioned, and the course is a typical one. Glaucoma has been observed after all methods of operation (even after division of secondary cataract). The author believes, however, that the disease occurs more frequently after extraction without iridectomy. Undoubtedly there is a greater predisposition to secondary glaucoma after extraction without iridectomy, on account of the more numerous accidents.

HIRSCHMANN.

XV.—SYMPATHETIC OPHTHALMIA.

146. ABADIE, CH. Pathogénie et nouveau traitement de l'ophtalmie sympathique. *Ann. d'ocul.*, vol. ciii., p. 183.

147. BASEVI. Patogenesi microbica della oftalmia migratrice. *Ann. di ottalm.*, vol. xix., 1, p. 57.

148. COPPEZ. Clinique ophtalmique de l'hôpital St. Jean. *Rev. gén. d'opht.*, 1890, p. 97.

149. GAYET. Recherches anatomiques sur une ophtalmie sympathique expérimentale. *Arch. d'opht.*, vol. x., No. 2, p. 97.

150. GUAITA. Studio anatomico clinico sulla exenterazione del globo oculare. *Ann. di ottalm.*, vol. xix., 1, p. 3.

151. OHLEMANN, M. Perforating injuries of the eyes with reference to the occurrence of sympathetic opthalmia. *Arch. f. Augenhk.*, vol. xxii., p. 94.

ABADIE (146) has endeavored to treat sympathetic ophthalmia with injections of sublimate. He obtained good results with this method in three cases, which he reports. He injects two drops of a solution of sublimate (1:1000) into the injured eye, and when the other eye is also severely affected he likewise injects a drop of the same solution. MARCKWORT.

BASEVI (147) has seen a severe reaction, and in two cases death of the animal experimented upon, follow inoculation of the anterior chamber or vitreous with pure cultures of staphylococcus, but never the development of sympathetic ophthalmia in the other eye. He found, however, a small, moderately mobile bacillus, besides streptococci and staphylococci in the tissues, and especially in the optic nerve of enucleated eyes, which had caused sympathetic ophthalmia. Pure cultures of this bacillus on agar or gelatine, and introduced into the vitreous or optic nerve of a healthy eye, produced chronic irido-cyclitis, and about a week later sympathetic lesion of the other eye. In twenty experiments on rabbits, the primary reaction developed in sixteen and the sympathetic in eleven cases. In five carefully examined human eyeballs, which had been enucleated on account of sympathetic inflammation of the other eye, the author found this bacillus, together with strepto- and staphylococci, and in especially large numbers in the region of the ciliary body. DANTONE.

COPPEZ (148) ably and energetically defends enucleation for sympathetic ophthalmia against Wecker's attacks. (*Cf.* de Wecker, L'abus de l'énucléation, *Ann. d'ocul.*, vol. cii., p. 192.) MARCKWORT.

GAYET (149) inoculated the left eye of a rabbit with spongy granulations taken from a lachrymal sac, and saw iritis develop after a month, and two weeks later a severe inflammation resembling sympathetic ophthalmia in the other eye. He found the lens infiltrated with round cells, the retina detached, interstitial neuritis of the left optic nerve, the left half of the chiasm sclerosed, and without a trace of nerve fibres. In the right optic nerve he also found interstitial neuritis, which, however, did not reach the globe. In the second eye he mainly found sclero-keratitis, with formation of an abscess and vascularity of the cornea. Gayet thinks the deleterious substances were first carried through the optic nerve to the chiasm, and then to the other eye, leaving the optic nerve, however, at the entrance of the central blood-vessels, and reaching the corneal margin with the subdural lymph-stream. v. MITTELSTÄDT.

GUAITA (150) comes to the following conclusions in regard to exenteration: 1. Enucleation is absolutely indicated in malignant tumors and sympathetic ophthalmia; exenteration in panophthalmitis and retro-ocular suppuration. 2. Enucleation is preferable in painful phthisis, exenteration in hydrophthalmic degeneration, total corneal staphyloma, absolute and hemorrhagic glaucoma. He uses no sutures whatever, removes the contents of the globe with a disinfected sponge, cauterizes superficially with the red-hot iron, inserts merely a strip of gauze for drainage, makes cold applications with a sublimate solution (1 : 1000), and of course observes strict antiseptic precautions. DANTONE.

XVI.—LENS.

152. CICARDI. Due casi di sublussazione della lente cristallina. *Ann. di ottalm.*, vol. xviii., 6, p. 548.

153. ROBERTS, JOHN B. Extraction of cataract. *Med. Progress*, Jan., 1890.

154. SCHIRMER, O. Supplementary remarks on the pathological anatomy of zonular cataract. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 1, p 135.

155. SWANZY, H. R. Remarks on a series of one hundred cataract extractions. *Brit. Med. Jour.*, March, 1890, p. 531.

156. TROUSSEAU. Les maladies générales et l'opération de la cataracte. *Soc. d'opht.*, 1890, p. 145.

CICARDI (152) describes two cases of partial traumatic dislocation of the lens, in which he succeeded in restoring the lens to its normal position with the aid of eserine, compressive bandage, and rest. At the first examination of both patients he found a myopia of 5 D, which finally disappeared almost entirely, while sight rose to $\frac{20}{40}$ and $\frac{20}{20}$ respectively. DANTONE.

ROBERTS (153) uses the customary aseptic precautions. He makes the upward incision and an iridectomy. His dressing is a semi-ellipse of plaster applied to the upper lid alone, holding it stiff like a splint, the lower lid remaining free. No bandage is applied, and the patient is allowed much freedom of movement. Thirteen cases are reported in more or less detail, all successful.
BURNETT.

SCHIRMER (154) proves with the microscope that the globules, which make up the opaque layer in zonular cataract and also penetrate into the nucleus, are present during life, and are not the result of hardening in alcohol.

SWANZY (155) operates by the "3-*mm* flap" method, observing strict antiseptic precautions. Iridectomy is always performed, the capsule freely divided, lately by transverse scorings only. The cortex is removed by the lid manœuvre. The author makes a point of searching the corneal wound with an iris forceps, in order to remove any bits of capsule which may have lodged in it. Tags of capsule had to be removed in nine cases. Good sight was obtained in 93 %, moderate in 5 %, loss 2 %. In the discussion of the merits of operating with or without an iridectomy, the size and position of the coloboma is constantly left out of view. The author makes the iridectomy above, and from 2 to 4 *mm* wide in the centre. That the average acuteness of vision is higher in simple extraction requires proof. The high average obtained in Professor Knapp's last report is rather due to the number of discissions, 74, as against 21 performed by the author. WERNER.

TROUSSEAU (156) believes that in operating for cataract in cases of diabetes and albuminuria there is no special cause for apprehension. He thinks the chances of success are smallest in patients with cerebral affections and in topers.

MARCKWORT.

XVII.—VITREOUS.

157. HILL-GRIFFITH. The artificial vitreous body. *Brit. Med. Jour.*, Feb., 1890, p. 293.

158. HARLAN, G. C. Extensive vascular growth in vitreous. *Trans. Amer. Ophth. Soc.*, 1889.

HILL-GRIFFITH (157) has performed Mules' operation 11 times up to the end of 1886, and in 2 only the glass sphere came out; one of these was considered a very satisfactory example of the completed operation. The author has not found that a drain of any kind has mitigated the severe reaction which follows; he uses thick silk sutures passed through conjunctiva and sclerotic. The entire operation takes ten minutes. WERNER.

HARLAN'S (158) case is accompanied with a good figure, which shows a triangular network of delicate blood-vessels extending into the vitreous from the optic disc, but not originating from the retinal vessels. Vision was fairly good. As there were hemorrhages Harlan thinks these vessels may have originated in them. The vessels were disappearing at the last examination.

BURNETT.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

159. AYRES, S. C. Retinitis pigmentosa treated by injections of strychnine. *Amer. Jour. Ophth.*, Oct., 1889.

160. Berry. Note on a stereoscopic effect and its application. *Ophth. Rev.*, vol. ix., p. 103.

161. Darier, A. Dégénérescence cystoïde bilatérale de la rétine à évolution lente et progressive. *Arch. d'opht.*, vol. x., No. 3, p. 203. With illustrations.

162. Dommartin, Cécité subite consécutive à un traumatisme de la région occipitale. *Arch. de méd. et de ph. milit.*, 1890, No. 3, p. 211.

163. Harlan, G. C. Hysterical blindness of ten years' duration in a healthy male subject. *Trans. Amer. Ophth. Soc.*, 1890.

164. Hoffmann, J. Experiences on traumatic neurosis. *Berliner klin. Wochenschr.*, 1890, p. 655.

165. Logetschnikow, S. Cases of cure of detachment of the retina. *Wjestnik Ophth.*, 1890, No. 2.

166. Morton, H. S. Obstructed retinal circulation. *Ophth. Rev.*, vol. ix., p. 65.

167. Osten-Sacken, Leo von der. The progressive peripheral pulse of the retinal veins. Dorpat, Schnakenburg, 1890.

168. Probsting. A case of retinitis proliferans. *Zeh. klin. Monatsbl.*, vol. xxviii., p. 73. Can find no definite cause.

169. Ritter, J. Contribution to the knowledge of traumatic neurosis. *Berliner klin. Wochenschr.*, 1890, p. 361.

170. Ritzmann, E. The hypnotic suggestive treatment in diseases of the eye. *Correspondenzbl. f. Schweizer Aertze*, 1890, p. 79.

171. Schleich. Contribution to the knowledge of the spontaneous development of connective tissue in the retina and vitreous (retinitis proliferans [Manz]). *Zeh. klin. Monatsbl. f. Augenheilk.*, vol. xxviii., p. 63. He attributes it to hemorrhages into the retina and vitreous.

172. Uhthoff, W. Contribution to hemeralopia and xerosis conjunctivæ epithelialis. *Berliner klin. Wochenschr.*, 1890, p. 630.

Ayres' (159) case was that of a girl of eleven years who had the typical retinal picture of retinitis pigmentosa with a visual field of only 10°. After injections of strychnine daily for thirteen days it increased gradually to 30° downward, 40° outward, 22° inward, and 20° upward. Central vision also improved.

Burnett.

In Berry's (160) stereoscopic pictures, each eye has presented to it two concentric circles, the smaller central one being capable of lateral movement. If binocular vision exists, the effect appears to be that the central ring approaches or recedes from the observer; if not, the lateral movement alone is seen. WERNER.

DARIER (161) observed for four years some peculiar changes in the right eye of a woman aged thirty-three, whose left eye had been removed ten years previously. There was a whitish projecting spot downward and outward from the papilla, from which a delicate network developed into the vitreous, while the fundus was gradually converted into a white, nodular surface, with blood-vessels running over it. V finally sank to $\frac{1}{\infty}$. Microscopic examination of the left eye showed cystoid degeneration of the retina, and the affection of the right was probably of a similar nature. V. MITTELSTÄDT.

DOMMARTIN (162) saw the following interesting case: A workman aged fifty-five received a powerful blow on the back of the neck, was unconscious for twenty-four hours and blind on awakening. The appearance of the fundus was normal and the pupillary action preserved. After some time sight returned. Dommartin thinks that a hemorrhage had taken place which had compressed the cortical centres. MARCKWORT.

In HARLAN'S (163) case, that of a man of twenty-two years, with no history of any nervous derangement of any kind, there was a supposed blindness of one eye, which, however, under the tests was shown capable of vision. He was finally convinced that he could see and thereafter did see. The delusion had existed for ten years. BURNETT.

Among twenty-four cases examined by HOFFMANN (164) six exaggerated their symptoms and eight were simulants. The majority of authors have declared simulation of the whole well-known clinical picture impossible. Only ten of the twenty-four were tested in regard to the visual field, which was found contracted in three and of normal extent in seven cases.

In a case of unquestioned detachment of the retina with iridodialysis and hemorrhage, the result of a blow with a whip, LOGETSCHINKOW (165) saw recovery take place in nine days after instillation of atropine. HIRSCHMANN.

MORTON (166) considers that the arterial tension should be systematically examined, in case of obstructed retinal circulation. He records two cases presenting appearances of embolism or thrombosis associated with high arterial tension; in one there were albuminuria and aortic regurgitation, in the other only slight hypertrophy of the heart. A third case is described of obstruction of the retinal vessels occurring after several paroxysmal failures of sight accompanied with migraine, a condition in which high arterial tension exists, as shown recently by Dr. A. Haig.

WERNER.

OSTEN-SACKEN (167) concludes that the physiological venous pulse dependent upon the pressure in front of and behind the lamina cribrosa is aided by the variations in pressure in the cavernous sinus. In insufficiency of the aorta, in 30 % of all cases of general arterio-sclerosis, but not in valvular disease of the heart, and in many cases of anæmia and chlorosis, the pathological peripheric progressive venous pulse is found. He thinks it is due to the fact that the arterial pulse extends farther than under normal conditions and through the capillaries into the veins.

One of RITTER'S (169) cases is remarkable from the fact that only the organs of sense, sight, hearing, smell, and taste were affected. There was also paresis of the facial and hypoglossus nerves.

RITZMANN (170) reports the cases of two women suffering with blepharospasm and anæsthesia of the retina, which did not yield to the usual methods of treatment, but quickly to hypnotic influence.

UHTHOFF (172) presents an extensive review of the literature of our knowledge of hemeralopia and xerosis, and also attributes it mainly to strong sunlight and imperfect nourishment. He thinks he has found another cause in chronic alcoholism. Among 1,500 demented men, one third of whom were topers, there were 27 cases of hemeralopia.

XIX.—OPTIC NERVE.

173. CIRINCIONE. Tuberculosi del nervo ottico. *Giorn. di neuropatologia*, vol. vii., 2.

174. MACNAMARA, N. C. Rheumatic neuritis and neuro-retinitis. *Brit. Med. Journ.*, May, 1890, p. 1007.

175. PHILLIPS, T. Reports of cases. *Royal London Ophth. Hosp. Rep.*, vol. xii., 4, p. 331.

176. SCHEFFELS, O. On resection of the optic nerve. *Zeh. klin. Monatsbl.*, vol. xxviii., p. 197.

177. ULRICH, R. A case of uniocular incipient choked disc, with bilateral partial atrophy of the trunk of the optic nerve. *Arch. f. Augenhk.*, vol. xxii., p. 32.

CIRINCIONE (173) describes a tumor 14 × 18 *mm* which surrounded the optic nerve in the region of the posterior attachments of the ocular muscles. Careful examination under the microscope proved it to be a genuine tuberculous tumor, which penetrated the optic nerve and its sheaths, and had undergone partial fatty degeneration. The eye was taken from a girl aged eleven, who had died of basilar meningitis, and had been set aside for examination on account of extensive ectropium, hypertrophy of the lachrymal gland, and numerous corneal opacities. The author mentions that only three cases of tuberculosis of the optic nerve have been reported. In two of them (Cruveilhier and Hjort) the intracranial, and in one (Sattler) the orbital part of the nerve was affected. DANTONE.

MACNAMARA (174) records particulars of two cases of neuro-retinitis confined to one eye in young individuals with history of rheumatism. In both cases the attack was acute, the eye being very painful, especially on movement or on pressure. $V = \frac{1}{8}$, but there was great photophobia. Under anti-rheumatic treatment vision rose in one case to 0.6 Snellen, and in the other to Snellen 5. The discs remained white. The author also refers to ulcers of the cornea connected with rheumatism. WERNER.

PHILLIPS (175) records the case of a girl, æt. eighteen, who for five months was subject to optic neuritis at repeated intervals. Iodide of potassium and cod-liver oil always produced a beneficial effect. In the second case, a man, æt. thirty-four, got a blow on the head with a piece of iron ; no open wound ; was unconscious for three weeks. There was a depressed fracture over the posterior superior angle of the left parietal bone. $V = \frac{20}{200}$. Well-marked right lateral hemianopsia. There appears from the charts to be some contraction of the rest of the field, especially in the left eye. WERNER.

SCHEFFELS (176) reports on forty-one resections performed at the Wiesbaden Ophthalmic Hospital during the last seven years, and concludes from the clinical experience that resection performed correctly and at the right time protects against sympathetic ophthalmia. The operation was performed in fifteen cases in which the patients were suffering directly and exclusively from the blind eye, and in twenty-six in which sympathetic ophthalmia was feared. Pagenstecher attaches the divided muscle to the sclera. Finally he opposes Deutschmann's well-known experiments and the conclusions drawn therefrom.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

178. Collins. Curator's pathological notes. *Royal London Ophth. Hosp. Rep.*, vol. xii., p. 334.

179. Dujardin. Blessure de l'œil. *Jour. des sci. méd. de Lille*, 1889, No. 26, p. 601.

180. Fischer, E. Extraction of chips of iron from the vitreous. *Arch. f. Augenhk.*, vol. xxii., p. 18.

181. Fumagalli. Corpo straniero nell' occhio sinistro. *Ann. di ottalm.*, vol. xviii., 6, p. 544.

182. Hutchinson, J., Jr. Unusual cases of injury to the eye and orbit. *Royal London Ophth. Hosp. Rep.*, vol. xii., 4, p. 305.

183. Magawly. Cysticercus in the eye. *Wjestnik. Ophth.*, 1890, No. 1.

184. Sattler. On injuries to the eyes. *Prager med. Wochenschr.*, 1890, 1.

185. Schirmer. Two cases of rare injury to the eyes. *Deutsche med. Wochenschr.*, 1890, p. 457.

186. Vossius, A. An operation for cysticercus. *Berliner klin. Wochenschr.*, 1890, p. 7.

187. Wadsworth, O. F. Two cases of extraction of steel from the vitreous which had passed through the lens. *Trans. Am. Ophth. Soc.*, 1889.

Collins (178) describes the following conditions: (1) Dragging forward of the retina in the region of the ora serrata without general detachment; (2) three cases in which a foreign body, after entering the eye, rebounded from the back of the globe; (3) hernia of the lens through a perforation in the cornea, after rupture of an ulcer, and also after Saemisch's incision; (4) rupture of the globe, with displacement of the lens. After enumerating several other cases, the author recommends non-interference with a lens dislocated under the conjunctiva, if there be no pain.

Werner.

Fumagalli (181) saw a case of foreign body (piece of gun-cap in an atrophic eyeball), which had caused sympathetic ophthalmia attended with the most severe general symptoms, and even epileptiform convulsions. After enucleation of the eyeball the symptoms rapidly disappeared. DANTONE.

HUTCHINSON (182) records the following cases amongst others: A tin tack penetrating the eye and lodging in the vitreous humor; brick dust filling up half of the anterior chamber; a slate-pencil almost two inches long embedded in the orbit for several weeks, the eye escaping injury. Finally a case of complete removal of lens and iris, by a blow of a pipe-stem or piece of iron, with retention of $V = \frac{20}{50}$ with $+ 13$ D. WERNER.

MAGAWLY (183) publishes a case of successful extraction of a cysticercus from the vitreous; intraocular cysticerci, as well as the tænia itself, being a very rare occurrence in Russia. (The reviewer saw one case among 130,000 patients.)

HIRSCHMANN.

SATTLER'S (184) paper principally considers the complications in non-perforating injuries to the globe, such as rents of the sphincter, iridodialysis, opacities of the lens, rents of the capsule, dislocations of the lens, ruptures of the choroid, etc. In perforating injuries the principal indication is the prevention or treatment respectively of infection, as sympathetic ophthalmia frequently follows.

SCHIRMER (185) saw a case of rupture of the anterior capsule of the lens with an uninjured cornea, and a triple rupture of the sphincter of the iris. In regard to the cause of the latter, he is of the opinion that the iris is caught between the cornea and lens under the influence of the trauma, whereupon the free edge tears, as it cannot escape.

VOSSIUS (186) succeeded in extracting a cysticercus from the vitreous. Four weeks after the operation, $V = \frac{1}{6}$. He then describes the technique of the operation, and reviews the frequency of the occurrence of the parasite.

The first case of WADSWORTH (187) was that of a boy, of thirteen, into whose vitreous a small piece of steel had entered through the centre of the cornea and lens. The body was easily seen lying just behind the lens, which remained quite clear, and was extracted with an electro-magnet through a scleral incision. No reaction followed, but a complete cataract was the final result. He was killed by a railway accident before any further interference was instituted. Case 2 was a man of thirty-one years. A bit of steel entered the vitreous through the lower part of the cornea, iris, and lens. It was removed through a scleral incision with an electro-magnet. He did well for two months, when internal inflammation, accompanied with deposits of pigment on the disc and elsewhere, set in, and it finally resulted in a detachment of the retina, which however did not begin at the point of the scleral incision. The lens did not become wholly opaque.

BURNETT.

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

188. ADLER, A. On affections of the eyes due to influenza. *Wiener med. Wochenschr.*, 1891, p. 141.

189. ADLER, A. A case of subcortical alexia (also called optical aphasia). *Berliner klin. Wochenschr.*, 1890, p. 357. The affection is probably due to a destruction in the left occipital lobe and gyrus angularis.

190. BADAL and A. FAGE. Complications oculaires de la grippe. *Arch. d'opht.*, vol. x., No. 2, March-April, 1890, p. 136.

191. BERLIN. Purulent iridochoroiditis after influenza. Paper read before the Society of Rostock Physicians, March 8, 1890. *Correspondenzbl. d. allg. Mecklenburg Aerzte-Vereins*, 126, p. 282.

192. BOCK, E. Ophthalmic and aural experiences during an epidemic of influenza. Paper read before the Society of Physicians at Krain, Feb. 26, 1890.

193. BONO. L'oculistica e la medicina in generale. *Gazz. med. di Torino*, vol. xli., 14, p. 345.

194. VAN DEN BERGH. Un cas de métastase oculaire dans l'infection puerpérale ; énucléation ; guérison. *La Clinique*, 1889, p. 513.

195. BULL, C. S. Case of double choked disc, due to intracranial tumor, with autopsy. *Trans. Amer. Ophth. Soc.*, 1889.

196. COHN, S. Uterus and eye. Description of the functions and diseases of the female sexual organs and their pathological effect on sight. Wiesbaden, Bergmann, 1890.

197. DENTI. L'influenza e le malattie oculari. *Ann. di ottalm.*, vol. xix., 1, p. 77.

198. DAMSCH, O. On pupillary unrest (hippus) in diseases of the central nervous system. *Neurol. Centralbl.*, 1890, p. 258.

199. EVERSBUSCH. On the ocular affections occurring in influenza. *Münchener med. Wochenschr.*, 1890, p. 89.

200. FOERSTER. On cortical blindness. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 1, p. 94.

201. Groenouw, A. Five cases of paralysis of accommodation in meat-poisoning (ham). *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 166.

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203. Macnamara, A. C. Malarial neuritis and neuro-retinitis. *Brit. Med. Jour.*, March, 1890, p. 540.

204. Nathanson, A. Case of influenza with pleuro-pneumonia and binocular embolic irido-choroiditis. *Petersburger med. Wochenschr.*, 1890, p. 213.

205. Oliver, C. A. An analysis of some of the ocular symptoms observed in so-called general paresis. *Trans. Amer. Ophth. Soc.*, 1890.

206. Rampoldi. Affezioni oculari nell' corso dell' epidemia della "influenza." Nota clinica. *Ann. di ottalm.*, vol. xviii., 6, p. 515.

207. Rampoldi. Sopra alcune affezioni oculari in rapporte con la cosi della influenza. *Ann. di ottalm.*, vol. xix., 1, p. 70.

208. Remack, E. Basal hemianopsia. *Neurolog. Centralbl.*, 1890, p. 130.

209. Sédan. Contribution à l'étude des symptomes oculaires observés pendant l'épidémie d'influenza. *Rec. d'opht.*, 1890, p. 137.

210. Simi. Influenza. *Boll. d'ocul.*, vol. xii., 3, p. 2.

211. St. John, L. B. Case of hemianopsia with peculiar cerebral symptoms. *Trans. Amer. Ophth. Soc.*, 1889.

212. Sulzer, D. E. (of Winterthur). Troubles de la vision dans l'impaludisme. *Arch. d'opht.*, vol. x., 3, May–June, 1890, p. 195.

213. Wollenberg, R. Two cases of tumor of the posterior cranial fossa. *Arch. f. Psychiatr. u. Nervenkrankh.*, vol. xxi., 3, p. 778.

Adler (188) saw all kinds of eye-diseases after influenza, but finally comes to the conclusion that there is no form characteristic for influenza.

Badal and Fage (190) report on 30 cases of eye-disease observed at the university clinic at Bordeaux after influenza. In the majority of cases the conjunctiva, cornea, and lids were affected; in two there was iritis and in the same number irido-choroiditis, once with loss of the eye. One case of acute glaucoma is remarkable, another of paralysis of the superior rectus, and one of the external rectus, and one of amblyopia without any visible changes. The affections occurred both at the beginning and during the period of convalescence, and were especially severe in the latter. Diatheses, rheumatism, and scrofula, as well as previous ocular affections, favored their development. The paralyses are the same as those which occur after other infectious diseases, diphtheria and typhoid fever. The authors consider it probable that the microbes of the conjunctival sac, which in a healthy state of the body are inactive, can produce infection in constitutional diseases.

v. MITTELSTÄDT.

BULL'S (195) patient was a married woman of twenty-three, who suffered from vertex headache and double optic neuritis for more than a year, during the latter part of the time being blind, and with numerous indices of central brain lesion—hemianæsthesia, loss of smell, loss of memory, etc. A nodulated tumor was found involving the corpora quadrigemina, extending to the anterior crura of the cerebellum and into the left hemisphere and involving the thalamus and internal capsule. Forward it extended from the thalamus to the left occipital gyrus. The anterior corpora quadrigemina were dislocated forwards. The ventricles were enormously distended. The tumor was a glio-sarcoma.

BURNETT.

The contents of COHN'S (196) careful monograph are given in the title. For details the original must be consulted. The bibliography embraces 219 numbers, which refer to the connection between female diseases and affections of the eyes, making the book a valuable acquisition to any library.

The ocular affections observed by DENTI (197) during an influenza epidemic, the connections of which with the latter could be traced with certainty were : œdema of the lids, acute conjunctival catarrh, congestion of the conjunctiva with severe supraciliary pain, profuse lachrymation and contraction of the pupil, etc., also severe, certainly infectious, corneal ulcers, small, deep, frequently penetrating ulcers, congestion and inflammation of the iris and choroid with hypopyon, the outer membranes being uninjured, and opacities of the vitreous. In five of them there was increase of tension and slight mydriasis, so that an iridectomy became necessary. The author also saw several times asthenopia and paresis of accommodation, and in three cases distinct neuro-retinitis.

DANTONE.

DAMSCH (198) observed hippus, aside from affections of the eye itself, in changes in the chiasm with subsequent hemianopsia, in paralysis of the oculo-motor nerve, in nystagmus, in epilepsy, in the early stage of acute meningitis, but relatively most frequently in multiple sclerosis. He also found it in cerebral affections, in which there were simultaneously other symptoms of motor or sensory irritation. Into this category belong acute meningitis and circumscribed affections after apoplexies. In chorea there was no hippus. Neurasthenic patients showed it occasionally.

EVERSBUSCH (199) mentions keratitis dendritica, also a peculiar painful sensation in the orbit, which becomes especially severe after accommodative efforts, and which he ascribes to difficulty of contraction of the participating muscles, and which has an analogy in the pain in the muscles of the back and extremities. The affections of the conjunctiva and the œdemas of the lids do not differ from the usual catarrhal affections. He also saw cases of glaucoma, serous irido-choroiditis, purulent choroiditis, and weakness of accommodation.

FOERSTER (200) saw a post-office secretary, aged forty-four, without syphilitic taint, who came to him in 1884 with an almost complete defect of the right halves of both visual fields. Vision gradually rose to $\frac{20}{30}$. In 1889 the left halves of the visual field were lost within a few days. The patient made the impression of a blind person. Closer examination, however, showed that a small central part on both sides retained its function with V = $\frac{1}{3}$. Colors could not be recognized. Foerster attributes the affection to thrombosis of the cortex. There were no symptoms of loss of function in any other part of the body. It was worthy of note that he had lost the power of picturing to himself the relative position of things in space. Foerster designates this as a loss of memory of place, and concludes that this faculty is dependent upon integrity of the occipital lobe. The fact that useful sight was preserved in a small area argues against the assumption that the macula lutea is supplied both by the crossed and uncrossed fibre-bundles. He sets up the hypothesis that, as the sensory-retinal elements always correspond to neighboring elements of the cortex, in case of thrombosis of the principal blood-vessel of the occipital lobe that part which corresponds to the macula lutea receives nourishment from another source, in consequence of numerous anastomoses, and therefore its function is preserved.

The case also shows that destruction of the cortex of the occipital does not cause atrophy of the optic nerve.

In patients, who had eaten a large quantity of ham, GROENOUW (201) found besides paralysis of accommodation moderate mydriasis and very sluggish pupillary reaction ; in others the pupils responded promptly. In regard to the differential diagnosis, it is important to note that marked mydriasis with slight paralysis of accommodation indicate the internal exhibition of atropine, and not meat poisoning.

HINSHELWOOD (202) records the case of a lad, aged seventeen, who fell down a ship's hold, receiving two scalp-wounds, but no fracture. He remained semi-comatose for two weeks, and the third week he was very irritable and easily excited. On the sixteenth day internal strabismus occurred from paralysis of the external rectus. Four days later the pupils were widely dilated, reacted on light, but not on convergence or accommodation. Patient could only read J 20, but with + 6 D, J 1. Distant V normal. Fundus normal. Patellar reflexes exaggerated. Accommodation recovered in eight days. WERNER.

MACNAMARA (203) records four cases of neuro-retinitis in patients who were subject to attacks of malarial fever. The defect of vision, which occurred during the attacks, and was due to neuro-retinitis, as shown by figures of the disc and tortuous veins, with minute hemorrhages, was cured with quinine and strychnine. The spleen was enlarged, there was no anæmia, no evidence of syphilis or rheumatism, no indication of serious renal mischief. The author is inclined to believe that the inflammation is due to the formation of ptomaines by microbes. WERNER.

NATHANSON'S (204) patient had had influenza in November, 1889. In the third week of January, 1890, pleuro-pneumonia set in, and on March 6th the left eye, and on the 8th the right eye, became blind.

OLIVER (205) has examined twenty cases of general paralysis—all men—in the second stage of the disease, and found evidences nearly always of lowered sensory nerve response on the part of the eye, and more or less implication of the ocular motor apparatus. The eyes generally showed evidences of wear and tear.

BURNETT.

RAMPOLDI (206) has observed the following conditions of the eyes attending and following influenza : Decided pain on movement of the globe, paresis of accommodation and paralysis, blepharospasm, congestion of the globe with increased lachrymation and photophobia, panophthalmitis from inflammation of old leucomas, increased tension even to an attack of glaucoma.

DANTONE.

RAMPOLDI (207) makes a second report on the ocular affections observed by himself in connection with influenza. Among 532 eye-patients whom he saw during the months of February and March, 1890, there were forty-five who had acquired their ocular trouble during or after an attack of influenza. These affections were: five cases of acute conjunctivitis, one of conjunctival abscess, five of abrasion of the corneal epithelium with anæsthesia of the spot, six of corneal infiltrations, one of corneal abscess, one of hypopyon-keratitis with acute irido-cyclitis, two of serpent ulcer, one of diffuse keratitis with serous iritis, one of simple serous iritis, one of glaucomatous irido-cyclitis, one of metastatic choroiditis (with loss of the eye), one of detachment of the retina in both eyes from serous choroiditis, six of supraorbital neuralgia, three of blepharospasm, two of spasmodic twitching of the lower lid, two of accommodative asthenopia, two of paralysis of accommodation, one of paralysis of the abducent, one of panophthalmitis of a previously healthy eye in a woman who had an adherent leucoma on the other eye. Among six cases of glaucoma there were two which most probably could be attributed to the prevailing epidemic. The experiments with the products of disease to find some special microbes failed. DANTONE.

The symptoms in REMACK'S (208) case were right-sided ptosis and paresis of the superior rectus, left-sided hemianopsia with partial atrophy of the optic nerve, and slight spastic paresis of the left lower extremity. In consideration of the circumstance that the limitation of the visual field had also affected the other half of the field, the focus of disease was referred to that part of the base where the tractus passes from above, behind, and outwards, downwards, inwards, and forwards across the pedunculus cerebri, and passes over the oculomotor, emerging from the latter.

SÉDAN (209) observed in a boy aged seven the certainly rare occurrence of blindness lasting two days during an attack of influenza. He was not consulted until after the blindness had disappeared, and could no longer find anything abnormal in the eyes.

MARCKWORT.

SIMI (210) saw in three persons, who had a choroidal affection and were attacked with influenza in the meantime, decided aggravation of the ocular disease. In four cases of simple and in one case of pustular conjunctivitis, which had developed during the influenza, he saw severe ciliary irritation, and in a sixth case during an influenza-pneumonia the formation of an atonic corneal ulcer, with perforation of the membrane and prolapse of the iris.

DANTONE.

ST. JOHN'S (211) case was that of a man who had had intense malaria, and who developed a nasal hemianopsia of both eyes—central vision being good—for which no cause could be found. The case was watched through five years. At last report central vision had declined to $\frac{15}{25}$ and $\frac{15}{64}$, the hemianopsia complete in the right eye, in the left less so; has feeling of pressure in the head, and is confined to bed.

BURNETT.

SULZER (212) presents a review of the ocular affections ascribed to malaria and adds a discussion of his experiences made in Java on soldiers of the Dutch colonial army. He saw most frequently optic neuritis in malarial cachexia. The frequent and marked variations in sight were characteristic. $V = \frac{1}{10}$ can rise in two to three weeks to $\frac{1}{2}$ or $\frac{2}{3}$, and in a few days go back again. In most cases the neuritis is binocular, but at first almost always confined to one eye. The field of vision is normal or concentrically contracted, color-perception normal, except in cases which end in atrophy. The dark coloration of the swollen papilla is also characteristic—a true melanosis. In 8 per cent. partial atrophy set in; in one third of the cases there were peripheric hemorrhages, which disappeared without a trace. Another manifestation of malaria is seen in the diffuse infiltration of the vitreous, which may become so intense that the papilla appears perfectly white. Atrophy of the latter is seen when the vitreous clears up, which, however, rarely takes place completely. Another variety of disease observed in the acute attacks, especially of febris tertiana in 20 per cent., consisted in a marked and even in the normal period persistent hyperæmia of the retina and the slightly swelled papilla. The choroid appeared as though covered wieh a delicate haze. The patients complain of severe stinging pain, photophobia, and flashes of light. This variety is inclined to develop into an affection of the macula lutea, similar to that observed after careless observation of an eclipse of the sun. The author found it in those malaria patients who came under his treatment on account of dazzling after long marches on the sandy coasts of Borneo, while otherwise such indirect dazzling did not lead to an affection of the macula. From the two latter forms the patients recover; chorio-retinitis only follows when malaria-cachexia supervenes. In conclusion the author sub-divides the affections as follows: (1) chronic optic neuritis; (2) diffuse infiltration of the vitreous; (3) peripheric punctate retinal hemorrhages; (4) sudden and permanent amaurosis. Acute attacks produce: (1) temporary amblyopia and amaurosis without any ophthalmoscopic changes; (2) diffuse affection of the vitreous with complication of the macula lutea; (3) extensive hemorrhages around the papilla and macula. v. MITTELSTÄDT.

In one of WOLLENBERG'S (213) cases there was a tumor of the temporal lobe which, besides the disturbances, had caused hemianopsia; in the other there was a tumor of the cerebellum with the following symptoms: disturbances of equilibrium, dizziness, headache, with stiffness of the neck. Early development of choked disc ending in amaurosis, marked nausea, but rarely vomiting. He also found limitation of the movements of the eyes, nystagmus, and symptoms of paralysis of facial and hypoglossal nerves.

MISCELLANEOUS NOTES.

Privat-docent Dr. UHTHOFF, of Berlin, has been appointed Professor of Ophthalmology at Marburg in place of Professor SCHMIDT-RIMPLER, who has been called to Goettingen.

Dr. K. MELLINGER has established himself at Basel as "privat-docent" for ophthalmology.

Privat-docent Dr. A. MAKLAKOW has been appointed Professor extraordinarius at Moscow.

Dr. J. MITVALSKY has established himself as "privat-docent" for ophthalmology at the Czech University at Prague.

Privat-docent Dr. BUNGE, in Halle, has been appointed Professor extraordinarius, and to replace Prof. A. GRAEFE at the University during the latter's illness.

Dr. FEUER has established himself at Budapest as "privat-docent" for ophthalmology.

Dr. EMIL GRÜNING has been appointed ophthalmic surgeon to the N. Y. Eye and Ear Infirmary, to fill the vacancy caused by the death of Dr. Cutter.

Extension of the New York Eye and Ear Infirmary, SECOND AVE., COR. 13TH ST. During the past five years the rapidly increasing numbers in attendance at the Infirmary have demanded increased facilities for treatment, but it was not until the early part of the year 1890 that any work in that direction was begun. Since that time a large addition has been made to the old building, which has also been remodelled. The whole now forms an institution of ample accommodation and proper equipment.

In the first place, whereas in former years in the Eye Department the space allotted for the reception and treatment of the vast numbers of walking cases that come here was limited to one or two rooms, wherein the surgeons and patients suffered much from the confined quarters ; to-day they have at their disposal large and airy accommodation in which each surgeon, having his own room and own facilities, can, with greater satisfaction, treat the cases allotted him. In like manner the surgeons of the ear and throat department suffered, while at present there are suitable spaces and conveniences for the carrying on of their special work.

The operating-room, in which over a thousand operations are yearly performed, has been thoroughly overhauled and put in a completely aseptic condition.

In order to carry out the same idea of antisepsis a special room has been set apart for an operating-room for the Ear Department, inasmuch as the majority of cases of this class upon which operations become necessary are of a purulent and contagious character. Through the benevolent munificence of a friend of the institution [1] a room, with ward attached, capable of containing six beds has been endowed as an operating-room and special ward for Throat cases.

The old wards, which in some cases were scarcely habitable, have undergone a most extensive change, and to them have been added large and airy ones, suitably arranged with all the latest appliances for the patients' health and comfort. With this increase in our hospital capacity ample provision can be made for cases of a special nature, while those of an infectious character can be properly isolated.

In addition to the above increased space a certain allotment has been made for the reception of special cases, who do not care to be placed in the larger wards with others about them but prefer more privacy and retirement, in the shape of smaller rooms upon a separate floor.

The total amount of available space for the maintenance of patients consists of about 20 rooms, containing in all ample accommodation for about 120 beds, of which about 50 are already in use.

The whole building has been heated in the latest approved manner, and the furnishing of the institution has been performed with the strictest regard for antiseptic cleanliness and good ventilation.

A. D. Harvey, M.D., House Surgeon.

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[1] Mrs. C. F. McLean, who gave $10,000 (Olliffe Memorial).

One of the features of the new arrangement is the LABORATORY, which is under the direction of Dr. John E. Weeks.

A light, well ventilated room has been set aside for laboratory purposes, newly fitted up with appliances for the study of the normal and pathological histology of the eye. A number of microtomes and a full line of apparatus for work in bacteriology have been supplied. There is also to be a room arranged for the purposes of micro-photography, the apparatus for which has been imported.

It is intended that the work in the laboratory will form an integral part of the work of the Infirmary. The microscopical examination of tissues removed by operation on all patients will be made here, and the results will be added to the histories of the cases.

It is proposed to preserve the specimens that come to hand, so as to form a valuable permanent collection, which will aid in the study of the various forms of eye disease.

Provision has been made for the accommodation of six students. Systematic instruction will be given by Dr. Weeks.

Besides some smaller legacies and donations (one of $2,000 from its old surgeon and generous patron, Dr. Abram Dubois), the Infirmary has received $25,000 from the Fayerweather estate, in the shape of five endowed beds at $5,000 each.

The late Mr. Fayerweather left in his will, $25,000 to the **Manhattan Eye and Ear Hospital**. The will was contested, but a settlement was effected and the Hospital is to get $50,000. Two other legacies, one of $3,000 and one of $1,000, have been left this Institution within a short time, the larger one by Mr. Thomas Hope.

Obituary.

Dr. V. F. SZOKALSKI,

whose name is frequently met with in the French literature of the period immediately before the invention of the ophthalmoscope, died January 6, 1891, at Warsaw. He was born in 1811, and made his studies in Warsaw, Heidelberg, Wûrtzburg, and Paris. He was chief of clinique of Sichel, and published many papers in the *Annales d'Oculistique*. His courses at the Ecole Pratique were well attended, among others also by A. v. Gräfe. Since 1853 he lived in his native country, at first in Cracow, then in Warsaw as Professor of Physiology, Opthalmology, and Otology. Among his contributions to Opthalmology we may mention those on the Corneal Leucomas, the Diphtheritic Ophthalmia, the Senile Visual Disturbances, the Functions of the Mucous Membranes of the Eye, and the Cerebral Visual Disturbances.

H. K.

Dr. MARTIN, OF MARSEILLES,

died in January, 1891. He was a fertile writer, well-known to every oculist who has followed modern ophthalmological literature. He founded an important Ophthalmic Clinque which will be conducted in the same spirit by his son-in-law, Dr. Changarnier.

H. K.

Dr. EDW. MICHAËLIS,

born in Berlin 1824; died there January 15, 1891. He was an assistant of v. Gräfe's from 1851–1862, and one of his most devoted friends. He helped in perpetuating v. Gräfe's memory by his monograph: "A. v. Gräfe, his Life and Work," 1877.

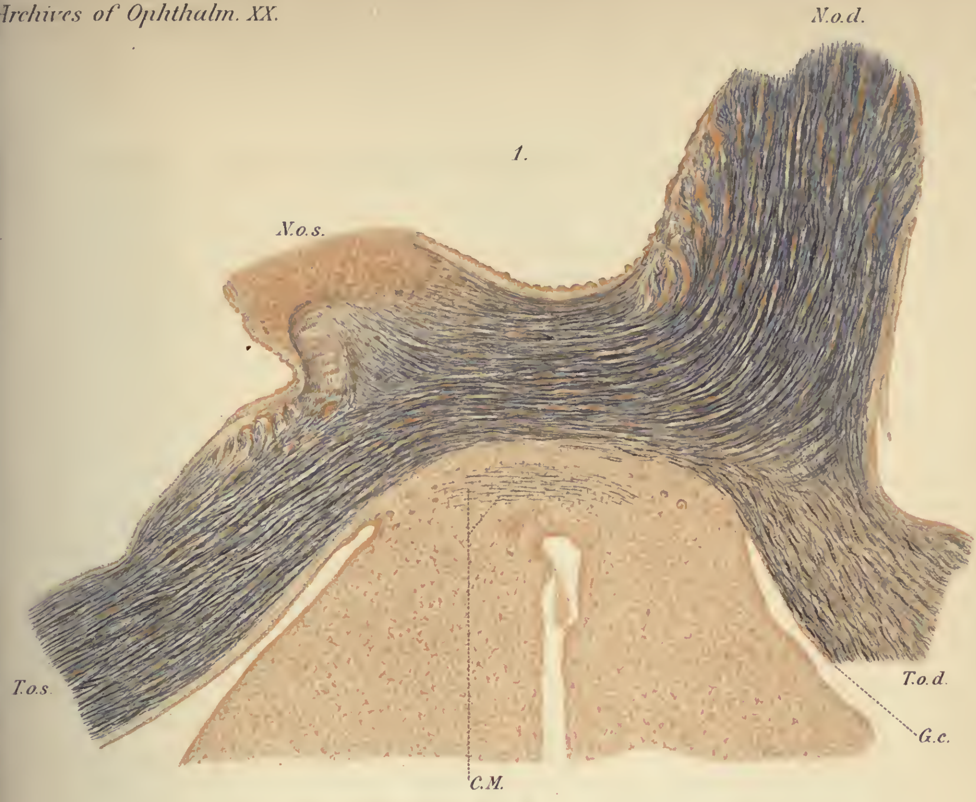
H. K.

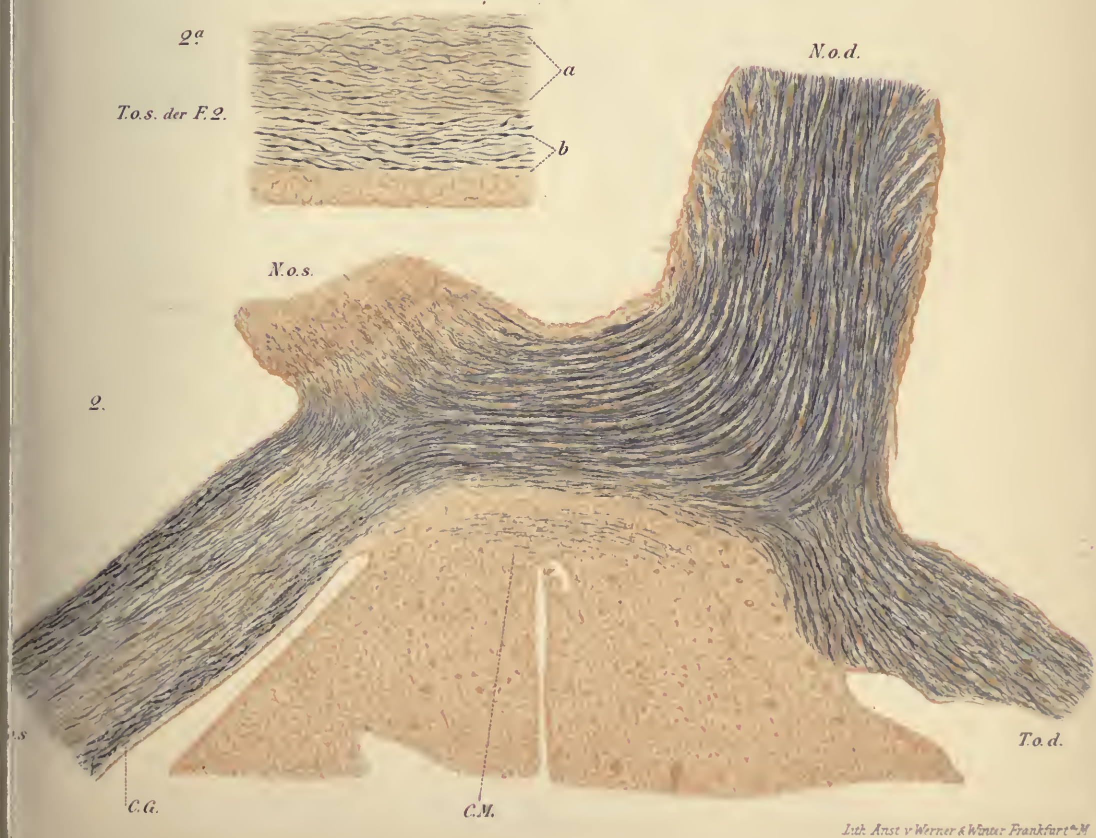
Dr. E. WARLOMONT

died at Brussels January 22, 1891, at the age of 70 years. Since 1850 he was the editor of the *Annales d'Oculistique.* He took up this journal in rather a precarious condition, but raised it to one of the most esteemed periodicals of its kind. His "elegant pen" was devoted more to comprehensive reviews and the diffusion of knowledge than to the promulgation of his own investigations; yet these comprehensive articles were not without a personal stamp. In 1857 he inaugurated at Brussels the first international Congress of Ophthalmology. The translation of Mackenzie's classical text-book into French, which he made in conjunction with Dr. Testelin, rendered a great service to French and German oculists not familiar with the English language. In the youth of modern ophthalmology, when, after Helmholtz's immortal invention ophthalmology took the lead in medical progress, he never failed at the Heidelberg meetings, and was a conspicuous personage even at the side of Gräfe, Donders, Arlt, Bowman, and Critchett. In one respect he fundamentally differed from his great colleagues. Devoting his time and energy to hospital patients, literary and scientific pursuits, he *hated private practice.* His vivacity, eloquence, and refined manners will never be forgotten by those who had the good fortune of making his personal acquaintance. H. K.

Dr. GEO. R. CUTTER,

since 1877 Surgeon to the N. Y. Eye and Ear Infirmary, died February 12, 1891. He was a gifted and scholarly man, faithful in the discharge of his duty, kind to his patients, and a competent practitioner. H. K.





Lith. Anst. v. Werner & Winter Frankfurt a/M.

Tab.XVII.

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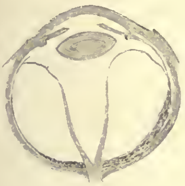

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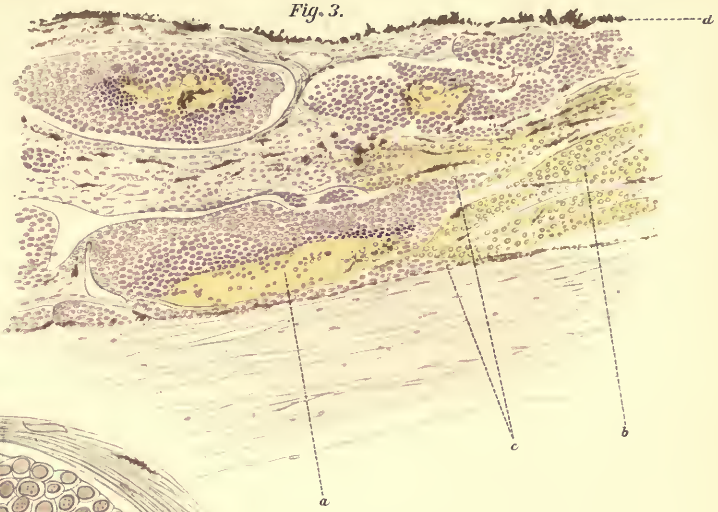

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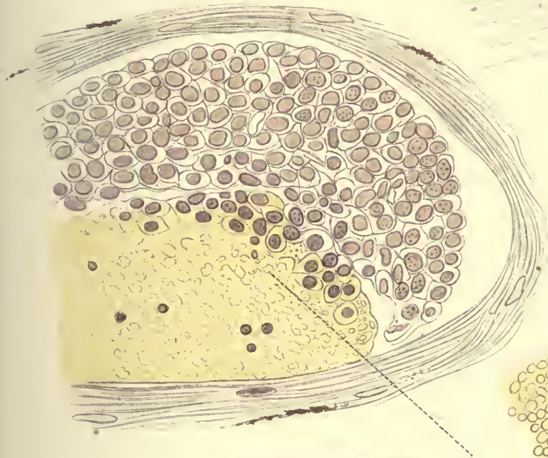

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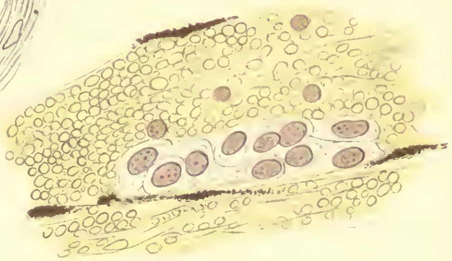

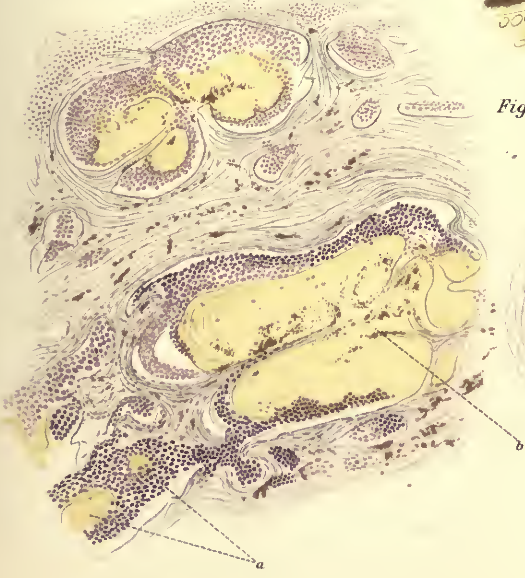

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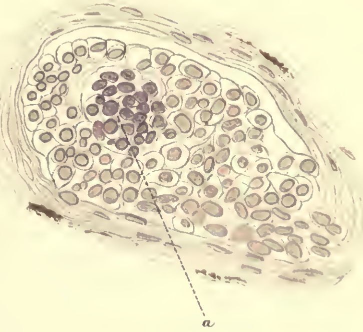


Lith. Anst. v. C. Kirst, Leipzig

Fig. 1.

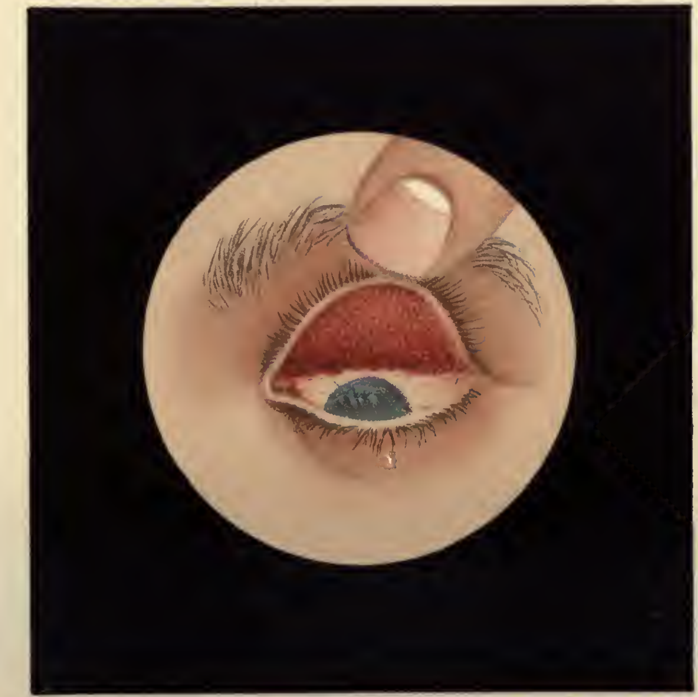
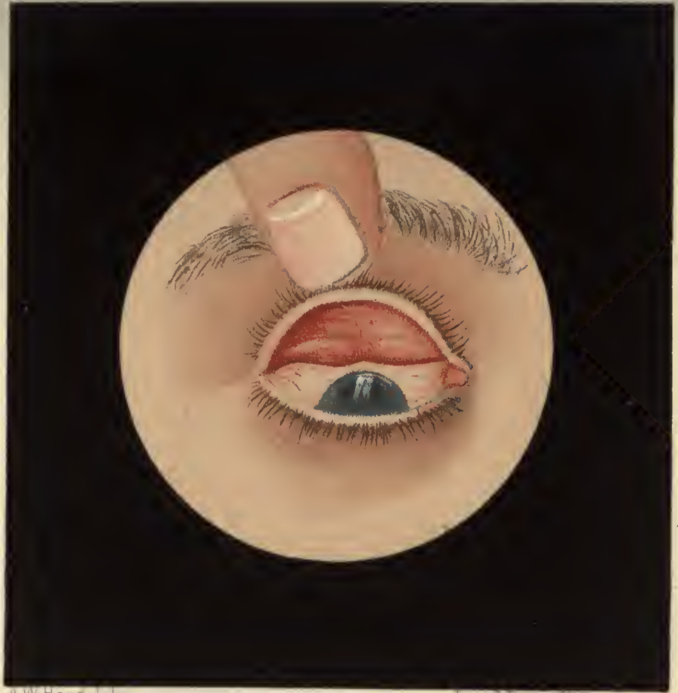


Fig. II.



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ARCHIVES OF OPHTHALMOLOGY

NOTICE TO OUR READERS.

The ARCHIVES OF OPHTHALMOLOGY contain *original papers* on all branches of ophthalmic surgery, and *original reports* on the progress of ophthalmology. The original papers occupy from two thirds to three fourths of the space, and their scope embraces all subjects of scientific and practical interest connected with this department of medicine.

The object of the ARCHIVES is to guard, solidify, extend, and diffuse the stock of acquired ophthalmic knowledge by recording the constant addition of new observations, investigations, methods and means of examination, clinical experience, instruments and remedies, and further by taking part in the discussion of the questions of the day with a spirit alike conservative and progressive.

Particular attention is paid to the preparation of the Report on the Progress of Ophthalmology. The report is intended to furnish a *complete*, *systematic*, *and early* review of the current ophthalmological literature of the world. It has been necessary to divide the work of its preparation among a number of collaborators according to the following arrangement :

Dr. ST. BERNHEIMER, of Heidelberg.

1. *General Ophthalmological Literature :*
   a.—Text-books (not abstracts, but notes pointing out what is new and characteristic).
   b.—Monographs and papers on historical, statistical, and general subjects ; reports on societies and hospitals.
   c.—Periodicals on our specialty (short notices of their character, time and place of publication, the titles of the original papers, with the names of their authors).

2. *General Pathology, Diagnosis, and Therapeutics*, including new instruments, apparatus, and remedies.
3. *Normal Anatomy and Physiology*, including color-blindness.

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2. *Eyelids.*
3. *Lachrymal Apparatus.*
4. *Muscles and Nerves.*
5. *Orbit and Adjacent Cavities.*
6. *Conjunctiva, Cornea, Sclerotic.*
7. *Injuries and Foreign Bodies of these parts.*

Dr. P. SILEX, Lecturer at the University of Berlin.

1. *Uveal Tract*, including glaucoma.
2. *Refracting Media*, including the lens.
3. *Retina and Optic Nerve.*
4. *Injuries and Foreign Bodies of these parts.*
5. *Ocular affections in connection with general diseases.*

In order to obtain complete and recent reviews on the non-German ophthalmological literature, special reporters have been selected for different countries, who will send monthly abstracts of all local ophthalmological publications to Dr. C. Horstmann, of Berlin, the editor of the Report. Dr. Horstmann will distribute these abstracts among the German reporters according to the above plan, in order to preserve the systematic arrangement which we think of paramount importance.

The special reporters are:

For America: Dr. SWAN M. BURNETT, 1770 Massachusetts Ave., Washington, D.C.

For Great Britain and Ireland: Dr. L. WERNER, 27 Upper Merrion Street, Dublin.

For France and Belgium: Drs. E. MARCKWORT, Antwerp, and P. VON MITTELSTÄDT, Metz.

For Germany: Drs. BERNHEIMER, Heidelberg, HORSTMANN, SILEX, and DEUS, in Berlin.

For Italy: Dr. DANTONE, Rome.

For Russia: Prof. HIRSCHMANN, Charkow.

For Scandinavia: Drs. OLE BULL and SCHIÖTZ, Christiania.

For Holland: Dr. C. A. H. WESTHOFF, of Amsterdam.

In the interest of our readers, who are scattered over the whole world—and whom, like ourselves, we desire to keep informed of all advances in ophthalmology,—furthermore in their own interest, authors of ophthalmological works are requested to send reprints, duplicates of journals, or copies for reviewing, to one of the above-named reporters, or to the editors or publishers of the Archives, according to their convenience.

The abstracts of American ophthalmological literature formerly appeared under a special head, but are now embodied in the systematic Report. Text-books may be specially noticed.

The number of good papers offered us for publication has increased to such an extent that it is no longer possible to translate all of them unabridged. Whenever a German paper is condensed in the English version, or *vice versa,* it will be so stated. Should any one of our readers wish to recur to the original, the editors will always take pleasure in sending it for reference.

Under the heading of "Miscellaneous Notes," there will be published all kinds of professional news which specially concern the oculist, *e. g.*, appointments, honors, resignations, vacancies, new ophthalmic hospitals, opportunities for instruction, prize questions and essays, announcement of Society meetings, etc. Brief notices of this kind will be thankfully received by the editors.

Original papers of value from any source will, as heretofore, be welcome to the ARCHIVES, and are solicited.

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NOTICE TO CONTRIBUTORS.

The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with their contributions.

1. As original communications the ARCHIVES can accept only such papers as have never been printed nor are intended to be printed in other journals. If a preliminary communication on the subject of a paper has been published, the author is requested to state this in the letter accompanying his manuscript. It is understood that contributors to these ARCHIVES and editors of other periodicals will make no abstracts of the original papers published in this journal without giving it due credit for the same.

2. Authors will receive gratuitously twenty-five reprints of their articles. If a greater number is desired,—notice of which should be given at the head of the manuscript,—only the additional cost of presswork and paper will be charged to the author.

3. In preparing manuscript for the compositor it is requested that the following rules be adhered to:

a. Write on one side of the paper only.

b. Write without breaks, *i. e.* do not begin a new sentence on a new line. When you want to begin a new line or paragraph at a given word, place before it in your MS. the sign ¶.

c. Draw a line along the margin of such paragraphs as should be printed in smaller type—for instance, all that is clinical history in reports of cases, etc.

d. Words to be printed in *italics*, should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times.

4. Authors may receive proofs for revision if they will kindly return them without delay. We beg however to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them, to make, if possible, no alterations at all in their MSS., or, at least, to limit these to what is of essential importance.

VOL. XX. No. 3

ARCHIVES OF OPHTHALMOLOGY.

REPORT OF FOUR HUNDRED AND FIFTY SIMPLE EXTRACTIONS OF SENILE CATARACT.

FROM PROF. SCHWEIGGER'S UNIVERSITY EYE CLINIC.

By Dr. RICHARD GREEF,
ASSISTANT SURGEON.

Translated by Dr. J. A. SPALDING, Portland, Me.

THE extraction of senile cataract without iridectomy has so far obtained less of a foothold in Germany than in France, England, and America; less, in fact, than it has actually deserved. Pagenstecher and Steffan have raised their voices against the new method, and Alfred Graefe[1] thinks "so long as it cannot be proved that the iridectomy interferes with the cicatrization of the incision, or subsequently injures the functions of the eyes, and so long as it is true that iridectomy prevents prolapse of iris, and reduces the number of incarcerations, then in my opinion every attempt to make a principle of the omission of an iridectomy must be regarded as a fatal and retrograde movement in operative ophthalmology."

The only answer to such and even heavier reproaches can come from those who have an extensive amount of material at their command. If statistics can only prove to us that the successes and the failures are alike or nearly so in both methods, the superfluity of the removal of a portion of the iris will be demonstrated and manifest in suitable cases.

For these reasons it may be worth the while to review the case books of this hospital under the care of a man who has shown himself one of the most ardent defenders of the new method.

---

[1] *v. Graefe's Archiv*, Bd. xxxiv., 3, p. 225.

The number of cases was verified from the books of the institution, and the figures include all the operations, even those performed by the assistants during the absence of Professor Schweigger. The sight was tested when the patient left the hospital, generally on the twentieth day, and only occasionally when it was plain that the turbulence of the patient had contributed to a poor result was the acuteness of sight tested at a later visit.

I would also emphasize the fact that all of the patients were from the University Hospital, not from the private hospital,—a fact deserving of notice in so delicate an operation as cataract extraction, because the behavior of the patients has a great deal to do with the results, better results being generally obtained in private patients than in the dirty and unruly inmates of general hospitals.

Schweigger reported the results of his first few cases of simple extraction at the Heidelberg Ophthalmological Congress in 1888 (Report, p. 135), and for the sake of completeness I will here recapitulate whatever is indispensable in his paper.

The operation was first done with Beer's triangular knife, with a downward flap entirely in the corneal tissue, and embracing half of the cornea, capsulotomy with the ordinary cystotome; eserine after completion of the operation. The first ten patients thus operated upon had been operated upon before in the other eye with an iridectomy.

The results at the time of discharge from the hospital were eight cases with vision between $\frac{1}{2}$ and $\frac{1}{6}$, making 80 % of successes. One patient had a prolapse of iris, abscised in a fortnight, with $S = \frac{1}{6}$. The other two patients were discharged with secondary cataract, both capable of improvement at a later date.

The large section of the old school is a necessity in the new operation. Nothing can be worse than too small a section. The broad knife was chosen for that reason as well as to make the section as smooth and as even as possible.

In the next thirteen cases the section was made upward, still in the cornea, and the cystotome still used. The results were between $\frac{1}{2}$ and $\frac{1}{6}$ in ten cases, $\frac{1}{18}$ in one case, which later by discision was raised to $\frac{1}{4}$. In one case there was prolapse of iris with $S = \frac{1}{6}$, and finally one case was lost from infection of the vitreous.

A following series of cases was operated upon with Schweigger-Foerster's capsular forceps, and these we will collect into one group in order to compare them with former statistics in which the same forceps were used, but with an iridectomy.

Beer's broad knife, which at a distance of 30 *mm* from the point is 4 *mm* high, was soon supplanted with one 5 *mm* high, and later still with one 7 *mm* high, a proof of the pressing advantages of a broad knife. The section was made upward in 117 cases. We first attempted to reduce the pressure on the eyeball to a minimum by holding the lids apart with the fingers, but soon found it disadvantageous to the operator. After trying various écarteurs we finally decided in favor of simple lid-holders. With these the assistant separates the lids (whilst the operator makes the incision), and thus is able to oppose or softly yield in a moderate degree to any pressure with the lids involuntarily exercised by the patient. The lid-holders are removed the moment that the section is completed. The lens is then removed whilst the eye is kept open by the fingers of the operator and his assistant.

We avoid tearing the conjunctiva during the operation by rejecting fixation forceps, merely using a two-tined fork to press, as it were, the eyeball against the knife during the completion of the section. But in avoiding the fixation forceps it seems as if the downward section be preferable, because anxious patients often roll the eyeball upward and endeavor to conceal it under the upper lid.

Twenty-five cases were operated on with the downward section and the capsular forceps, so that we get a series of 142 extractions in which the capsular forceps were used, with the following results:

Calculating $S = \frac{1}{6}$ as a good result, we have

Perfect success,	104 =	73.3 %
Secondary cataracts,	30 =	21.1 %
Losses,	8 =	5.6 %
	142	100 %

The vision obtained in the 142 cases was $\frac{6}{9}$ in 5 cases, $\frac{6}{12}$ in 19, $\frac{6}{18}$ in 38, $\frac{6}{24}$, in 22, and $\frac{6}{36}$ in 20 cases.

There was not a single case of $\frac{6}{6}$ at the time of discharge, owing to the greater or less amount of astigmatism which is present at an early stage, but gradually decreases or disappears. I am of the opinion that in order to get $S = 1$ in the statistics, one needs a little indulgence on the part of the physician, or even the use of various test type, and perhaps of hook letters. We have, however, found three cases noted at a later date as having $S = 1$, but the data are defective, and no actual claim of that amount shall be made in this paper.

Secondary operations were performed on 16 of the 30 capsular cataracts, all of which but one gained by the operation, and 8 above $S = \frac{1}{6}$, so that they are to be added to the good results. The cases which were discharged without any chance for improvement, or with preservation of a small amount of sight, are all included under the head of losses.

Two of the losses ought to be excluded because they were complicated, the one with irregular astigmatism from old leucomata of the cornea, the other with choroiditis at the macula.

The remaining six losses were due: one to hemorrhage into the anterior chamber directly after the operation, two to a low form of irido-choroiditis, secondary cataract, posterior synechiæ and thickening of the iris, $S = \frac{1}{200}$ and no chance for improvement; one to prolapse of iris with useful sight at first, then irido-cyclitis and amaurosis; three other cases were lost from infection of the section. If now we wish to put our statistics in a more favorable light, we shall, by omitting the complicated cases, have 140 operations resulting as follows:

Successes 104 + 8 (by discision), =	112 =	80 %.
Half successes	22 =	16.8 %.
Failures	6 =	4.2 %.
	140	100 %.

The cases of prolapse of iris are of great interest, nine in number, of which eight were abscised.

1. $S = \frac{6}{24}$.
2. $S = \frac{1}{9}$, later $\frac{6}{36}$, still later $\frac{6}{12}$.
3. $S = \frac{6}{12}$, later $\frac{6}{9}$, Schweigger's type 0.4 at 25 *cm.*
4. $S = \frac{1}{24}$, astigmatism ; no further notes.
5. $S = \frac{1}{12}$, later $\frac{1}{9}$.
6. $S = \frac{1}{12}$, later $\frac{1}{9}$.
7. $S = \frac{1}{6}$, later $\frac{6}{18}$.
8. $S = \frac{1}{12}$; macula of cornea.
9. $S = \frac{6}{18}$, later $\frac{6}{12}$.

Prolonged observation showed that five eyes obtained and preserved $\frac{1}{6}$ and even more ; one with macula of cornea is not counted, one with $S = \frac{1}{24}$ was not seen again, but it could surely be improved, and the last case obtained $\frac{1}{9}$ after discision.

This shows that the fate of eyes with prolapse of iris is by no means hopeless.

Although we are able to assert that the large majority of cases of prolapse have wonderfully improved and preserved their sight, yet we will not fail to mention that one case, after maintaining excellent sight for more than two years, finally succumbed to choroiditis with increased tension. It is, however, doubtful if the loss can correctly be ascribed to the original prolapse. The history of this case shall be reported farther on.

The xvi. vol. of these ARCHIVES, page 263, contains the statistics of 122 combined extractions at our clinic with the capsular forceps. The results were :

With iridectomy (February, 1883 to March, 1884).

Successes, . .	89 =	72.9 %.
Secondary cataract,	27 =	22.2 %.
Losses . . .	6 =	4.9 %.
	122	100 %.

Comparing these with the present simple extractions we have (1887–1888):

Successes . .	104 =	74.3 %.
Secondary cataract,	30 =	21.4 %.
Losses . . .	6 =	4.3 %.
	140	100 %.

Comparisons of the results of operations in different hospitals always hitch a little here and there. The way in which the operation is done, the operator's skill, the manner in which the statistics are made out,—all are different. And for that reason it is all the more interesting to compare the extractions in the same clinic, some with and others without the iridectomy, and then to find an agreement so general in every respect.

The next series of cases was operated with v. Graefe's cystotome, and a knife 7 *mm* high 30 *mm* from the point, in fact just such a wedge-shaped knife as is depicted in Zehender's Monatsblaetter (Band ii., p. 73), with a convex curve of the cutting edge. Up to the end of the year 1889 we performed 235 of these simple extractions with the section downward.

The results were as follows:

Complete successes,	185 =	79 %.
Secondary cataract,	39 =	16.2 %.
Losses . . .	11 =	4.8 %.
	235	100 %.

There were twenty cases of prolapse of the iris including the most minute.

1. The amount of sight regained was as follows:

$S = \frac{6}{9}$	10
$S = \frac{6}{12}$	32
$S = \frac{6}{18}$	54
$S = \frac{6}{24}$	48
$S = \frac{6}{36}$	41
	185

2. Some of the cases of secondary cataract may of themselves have obtained $S = \frac{1}{6}$, and in point of fact ten actually obtained this amount after a discision. Others were improved considerably.

One case was marked as a failure although when discharged $S = \frac{1}{100}$. Two additional cases of $S = \frac{1}{200}$ (occlusion of the pupil) were operated upon without success, so that these three last cases are included amongst the losses.

3. In place of the three cases which belong to the losses we have three others which were complicated.

CASE 1.—Mr. D., October 15, 1889. Right eye, operation smooth, pupil round, secondary cataract, $S = \frac{1}{36}$. Later discision, clear opening, $S = \frac{1}{24}$. Central scotoma for red and green ; patient smokes enormously. Suitable treatment was ordered, but the patient did not again report.

CASE 2.—Was about to have been operated on elsewhere a year before, but owing to incipient insanity was dismissed without the operation.

March 18, 1889, operation apparently successful, but the patient, who was rational in the early days subsequent to the operation, could not tell what he saw at the date of his discharge owing to a return of insanity.

CASE 3.—Macula of the cornea, $S = \frac{1}{12}$.

In one case, directly after opening the capsule the lens fell into the vitreous and could not be extracted even with the various hooks and spoons at our disposal. The cause of the luxation could not be discovered. S at time of dismissal $\frac{1}{5}$, but, as I learned by inquiry, the eye was slowly lost by irido-choroiditis.

Still another case was lost by hemorrhage into the anterior chamber.

Six eyes were lost from suppurative keratitis or hyalitis, with $S = \frac{1}{200}$ to 0. Amongst these, one eye was infected from a dacryocysto-blennorrhœa. Schweigger operates in these cases of lachrymal suppuration in the following manner, and this is the only case that he has lost from this cause in many years. On the day before the operation the lachrymal sac is split broadly open, and tamponed with sublimated cotton. After some hours, and when the hemorrhage has ceased, the tampon is removed, little rods of iodoform introduced, and then a sublimated tampon again applied. The iodoform rods are renewed once more previous to the operation, and after the operation they are changed daily.

A second case of infection was entirely due to the stupidity of the patient, who, in spite of the greatest care and watchfulness, repeatedly jumped out of bed, pulled off his bandages, and rubbed his eyes. The cause of the infection in the other cases was not discoverable.

It is very curious that in Prof. Schweigger's private hospital there has not been a single case of infection after extraction since the introduction of strict asepsis, whilst in the University Hospital, where precisely the same aseptic precautions have been scrupulously carried out, it has been impossible to avoid an occasional loss from infection of the corneal section. If aërial infection were not most positively denied by the most competent authorities, our first thoughts would run in this direction. Still, the hygienic conditions in the hospital leave much to be desired, although we hope to remove them soon by alterations in the building, whilst the neighborhood of the surgical clinic makes itself felt even in our wards.

All extractions now being regarded as dangerous operations, are no longer made in the large auditorium infected by the previous presence of a large number of persons, but in a smaller apartment and in the presence of but few spectators.

Our asepsis consists in the following procedures: On the evening before the operation, the skin around the eye to be operated upon is cleansed with ether, and then the eye as well as both the upper and lower everted eyelids are thoroughly laved with a 1 to 5,000 sublimate solution. The eye is then closed with a well-fitting pad of borated cotton, soaked in the sublimate solution. This remains till the moment fixed for the operation, when the conjunctiva is once more abundantly irrigated with the sublimate. The instruments are placed in a four-per-cent. solution of carbolic acid. It is well known that the eye will not tolerate so high a percentage, either of carbolic acid or sublimate, as is needed for disinfection, and for this reason we dilute this strength of acid on the instruments by a brief immersion in boiling water at the bedside of the patient.

It is an old experience, that the larger the clinic and the greater the number of attendants, the higher the percentage of suppuration. Amongst many hands, one is easily unclean, and here or there occurs the deadly sin of omission. Therefore we must try in every possible way to eliminate all those minute sources of failure and so perfect our method of operating.

Attention has already been called by various observers to the septic qualities of the cocaine solutions instilled into the eyes previous to the operation, and bacterial investigations have shown the masses of filth-bacteria that rapidly develop in such solutions. Actual boiling of the cocaine solution, just before the operation, as recommended by Eversbusch, must be useful. Only we must not forget that repeated boiling affects the strength of the cocaine by condensation of the water of solution. We may also recommend Sattler's method of preserving the cocaine in a sublimate solution of 1 to 10,000 or of 1 to 5,000, though Inouye has shown that the cocaine unites with the sublimate into an organic salt.

In order to avoid infection of the bandage with soiled hands I would also recommend that it as well as the cotton ought to be kept in one of Koch's steam sterilizers until the eye is ready to be covered, when both bandage and cotton can immediately be applied to the eye without being touched by the hand of any other person than the regular assistant. Since the opening of 1890 and the use of this method of bandaging, we have had only one case of suppurative keratitis after extraction, and that was undeniably due to the unruly behavior of the patient.

The first 50 cases of extraction this year resulted as follows:

Complete successes	36
Secondary cataract, astigmatism, etc. . .	12
Losses	2
	50

Of the second group, however, 7 soon obtained spontaneously or after discision, $S = \frac{1}{6}$. So that we really have these figures:

Complete successes . . .	45 =	90 %
Half successes	3 =	6 %
Failures	2 =	4 %
	50	100 %

There were four cases of prolapse of iris, one of which was not abscised. Two were complicated with secondary cataract. S amounted to $\frac{1}{9}$, later improving of itself to $\frac{6}{18}$;

$\frac{1}{12}$ improving to $\frac{1}{6}$; $\frac{6}{24}$ not seen again; and $\frac{1}{100}$ with secondary cataract to be divided later.

One of the *most important* chapters in any report on the extraction of cataract without iridectomy is the one on *prolapse of the iris.* This accident is indeed a sad occurrence, and one that cannot always be avoided. But the chief question is whether it occurs so frequently and is actually so serious an event as the opponents of the new operation would have us believe. The occurrence of the prolapse is generally followed by pain, which lasts for several hours, and by an irritated condition of the eye, which, however, generally yields in a short time to the application of warm fomentations. The prolapse should be treated as follows: if very small it may safely be left to itself, for it cicatrizes very rapidly, and leaves no trace behind except a trifling degree of astigmatism, which after a period of no great duration disappears, and forever. If the prolapse is of medium size, it *may* be left to itself, but it is on the whole better to abscise it in order sooner to get rid of the astigmatism which it invariably excites. We have abscised the prolapse as early as the eighth day. In about the same length of time the defect is covered over, and the patient can be discharged, so that actually he loses no time in the hospital.

We have not been able to discover that prolapse of iris is always associated with repeated attacks of iritis, or that iritis is more frequent after simple extraction than after extraction with iridectomy. We no longer have any faith, as Schweigger has expressed it, in an "antiphlogistic iridectomy." And more than that, incarceration of the iris and prolapse of the iris is by no means excluded in the extraction with iridectomy, only the piece removed cannot prolapse or become incarcerated.

There were 35 cases of prolapse of the iris (7.9% in 451 extractions), of which number 9 were so minute that they were not touched, the remainder were all abscised. I have not taken the trouble to write to these patients, to inquire after the condition of their eyes, for I long since convinced myself that the opinion of people generally in regard to their eyes and health is not to be trusted. And more than that, the demands of patients vary exceedingly; some cataract patients are dissatisfied with a subsequent vision of $\frac{6}{9}$. Contrarily, it is interesting and instructive to print the well observed and long-verified histories of patients with prolapse of the iris, for the purpose of demonstrating how eyes thus affected can improve, and remain good, for a length of time.

CASE 1.—L. H., teacher; simple extraction April 2, 1887; wedge-shaped knife, upward section embracing one half of the cornea; capsular forceps, easy extraction of lens; eserine. Pain on the next night; bandage changed without inspection of the eye.

April 5th.—A large prolapse of iris embracing one half of the section.

April 16th.—Cocaine and then abscision with scissors.

April 29th.—Slight corneal astigmatism. $S = \frac{6}{24}$ with $+ 3\frac{1}{2}$.

May 18th.—$S = \frac{6}{12}$ with $+ 3\frac{1}{2}$. Reads 0.4 with difficulty at 20 *cm* with $+ 2\frac{1}{4}$.

April 6, 1888.—$S = \frac{6}{9}$ with $+ 3\frac{1}{2}$.

CASE 2.—Mr. C. K., æt. seventy-five. Simple extraction left eye, November 3, 1887. Section downward with 7 *mm* knife; after removal of lens the remnants of sticky cortex demanded the frequent introduction of a spoon, but at last the pupil was clear; eserine; bandage.

Nov. 4th.—Patient complains of pain, slight swelling of lids; atropine between the lids without opening the eye, moist bandage.

Nov. 5th.—Pain; prolapse at inner angle of incision, enlarging day by day despite eserine, and finally abscised Nov. 19th.

Dec. 9th.—$+ 3\frac{1}{2}$ $S = \frac{6}{18}$.

Jan. 14, 1888.—$+ 3\frac{1}{4}$ $S = \frac{6}{18}$.

March 6th.—$+ 3\frac{1}{4}$ $S = \frac{6}{12}$; Sn. 0.4 at 20 *cm*.

CASE 3.—Mr. G., æt. sixty-five. June 4, 1889, simple extraction downward, left eye.

June 7th.—Patient out of bed without permission, and hit his head against furniture; large prolapse of iris; eserine used unsuccessfully.

June 14th.—Prolapse abscised.

July 1st.—Discharged. $+ 4$ $S = \frac{1}{12}$: $+ 4$ S $\supset$ $+$ c 10 $S = \frac{1}{9}$ and $\frac{1}{6}$ —.

August 4*th.*— + 4, S = $\frac{6}{24}$.
August 28*th.*— + 4, S = $\frac{6}{12}$ and $\frac{6}{9}$.
Sept. 10*th.*— + 4, S = $\frac{6}{9}$. + 2½, Sn. 0.3 at 20 *cm.*
May 26, 1890.— + 4, S = $\frac{6}{6}$.

Other favorable cases of this nature might be cited, but space forbids, and we shall rather here call attention to a case in which an eye with a prolapse was lost two years after the operation.

F. W., an overseer. Simple extraction on the right eye, April 22, 1887; on the third day, broad and extensive prolapse of iris, with iritis and pain. It was not abscised.

May 12*th.*—Discharged; eye comfortable, considerable astigmatism. S = $\frac{1}{12}$.

About two years later patient returned, reporting that until a week before he had seen well, but since then had suffered with severe pain and loss of sight. At upper corneal margin a large staphyloma of cornea and sclerotic; anterior chamber shallow, and pupil occluded. No perception of light. T + 1.

The eye was at once enucleated, and found full of pus.

Although it is our opinion that an excision of the iris can easily and safely be dispensed with in a large percentage of cases, yet there are *proper indications for the combined method.* In order to determine just what these are, we must determine the causes of prolapse of the iris and test from this point of view the various methods of operating, and after-treatment of extraction.

Injuries after the operation are the most frequent cause of prolapse, and in this class we must include many cases in which the patient will not acknowledge that he has received any injury. But who is to guarantee to us that the patient follows our directions, and rests quietly in bed? Over and over again, patients take advantage of the absence of the nurse, and leave their beds for some secret or fancied reason, in the very act of which, not seeing anything, they easily run against obstacles of various sorts. There are certain patients who will yield neither to argument nor persuasion, and hardly even to force. Thus we had a prolapse of iris in a patient who bent over toward the floor on the second day, after getting out of bed against the strictest orders. He was warned of his danger, and advised to take the greatest care when he had his second eye operated on. But on the second day after the second operation, we saw him in the darkened chamber, pushing with his thumbs through the bandage into the operated eye so violently that the section was sprung, and the iris prolapsed.

Where better care is taken, and the patients are more reasonable, as in the private clinics, prolapse of the iris is not often observed.

Traumatic prolapse of the iris through the section once already cicatrized is rare after the fourth day, and yet we once saw it in a rather full-blooded woman whilst bending over during a bath on the twelfth day.

Prolapse of the iris may additionally be due to violent efforts at stool after the operation, and for that reason we invariably administer a cathartic on the day before the operation, so as to produce at least one movement of the bowels, whilst afterward we keep the bowels quiet for two or three days, if necessary, with tr. opii.

Generally it is impossible to discover the precise cause of the accident; the operation goes off well, the patient is quiet afterward, and there is no injury. It is exceptional for a prolapse to occur on the first day or within a few hours after the operation. We can discover without inspecting the eye, and merely from what the patient says, that the prolapse is there, that it was accompanied with pain, which, however, disappeared in a few hours. So that in this accident three things are possible: that the prolapse is due to anatomical causes; secondly, to the manner in which the operation is performed; or thirdly, to the treatment of the patient directly after the operation.

1. Prolapse of vitreous is one of the chief anatomical conditions after which prolapse of iris may be expected, therefore wherever this accident is probable, as in high degrees of myopia, fluidity of the vitreous, etc., it is best to perform the combined operation, and remove a portion of the iris.

It is worth observing that the prolapse often occurs in both eyes, and if it has not been caused by the bad behavior of the patient, there cannot be any better reason than that in decrepit persons the relaxation of the sphincter of the iris mechanically favors the prolapse of the iris, especially when the section is made downward.

For this reason we have often iridectomized the second eye, when, without any apparent reason, there had been a large prolapse in the first eye operated upon without an iridectomy.

It occasionally happens, as in the following cases, that if anything goes wrong, it is imperative to do an iridectomy even after the extraction of the lens.

CASE 1.—R. S., saloon-keeper, æt. sixty-two, diabetic for years, but in fairly good health at present; nuclear cataract, right eye (not diabetic); left eye, $S = \frac{1}{36}$. Extraction, January 4, 1890, right eye. Section downward with broad knife; difficult extraction; some cortical fragments left behind, and removed with spoon. In the latter step the anterior chamber filled with a black emulsion originating from the degenerated iris pigment (Kamocki, these ARCHIVES, vol. xvii., p. 41), and spreading out all over the under surface of the cornea. The introduction of the spoon was followed with slight prolapse of vitreous. Iridectomy performed, and then most of the liquid stroked out of the anterior chamber. Moderate iritis followed; good recovery; slight secondary cataract, $S = \frac{1}{9}$.

CASE 2.—Mrs. K., æt. seventy-five, very decrepit; right cataract, mature; left, immature; $S = \frac{6}{36}$.

May 22d.—Extraction upward with broad knife, and then the iris bulged out and could not be replaced so as to remain. The lens was easily extracted, but even then the iris could not be replaced so as to stay. Broad iridectomy; bandage.

May 23d, although patient had been perfectly quiet and had received no injury, the bandage was full of blood, and again on the following day. The section was also wedged apart with a clot of blood.

June 16th, patient dismissed. S = 0.

This is one of *those excessively rare cases of choroidal hemorrhage after extraction of the lens*, and means of course, that the hemorrhage followed directly upon the completion of the section, and that it was this which so curiously and so obstinately bulged forward the iris and compelled the iridectomy.

CASE 3.—C. N., a workman, double mature cataract, always myopic, very clumsy, and constantly nips his eyelids together at every attempt to touch them.

March 27th, extraction of the right cataract, constant forcible nipping of the eyelids; lens violently extruded from the eye, the moment that the capsule was divided; vitreous followed. Iridectomy at once. Vitreous opacities at time of discharge. $S = \frac{1}{6}$.

2. Now in regard to the second point, the method in which the operation is performed, the question is whether prolapse of the iris is oftener seen after the upward or the downward section. So far as we are concerned, our statistics are of no use at all. For a short time after the section was removed into the lower margin of the cornea, the operations, for the sake of stricter antisepsis, were performed in a more carefully arranged apartment. The patients were then carried down stairs into their rooms. Therefore, if we find an increase of prolapses with the downward section, it may possibly be due to the subsequent removal of the patient.

To quote statistics:

139 cases were operated with the upward section, and there were 8 cases of prolapse of iris, = 6 %.

270 were operated upon with the downward section, and there were 23 prolapses, = 8.5 %.

The last series of 50 cases was operated upon in the patient's room, the bed for the operation standing next to the patient's bed, and the patient in the presence of the physician gently moved across from one to the other. The section was generally made downward, only upward when the patient was very tractable.

It would further be interesting to study whether it is always best to iridectomize whenever after the extraction of the lens the iris is with difficulty replaced, or, in other terms *shows a decided tendency to prolapse.* I doubt if the clinical history will support the affirmative side of this assertion, for even when a prolapse has appeared inevitable, it has not always ensued; generally its origin is enveloped in darkness, and when it does appear it is almost always unexpected. Knapp (these Archives, vol. xvii., p. 51) advises an iridectomy wherever there is a tendency to prolapse and it is difficult to replace the iris.

When simple extraction once more came forward for public reception, all operators used eserine more or less. We, too, did not fail to use it after the operation as well as on the evening of the same day. But we soon discovered, to our satisfaction at least, that there was no need of it, and for that reason we have not used it for some time. It does not prevent prolapse of the iris, whilst it undeniably favors the formation of posterior adhesions.

3. Much more care is needed in the first few hours after the new operation than the old, and most operators are agreed in avoiding the least movement of the patient after the extraction of the lens. The patient must be comfortably put to bed, and remain there as quietly as possible for three full days. The bandage is renewed on the second day, but the eye is not to be inspected. After the lids have been carefully cleansed with moist sublimated cotton, atropine is to be dropped into the inner corner of the closed eye, from whence it will easily make its way inward. The eye is not to be opened till the third or fourth day, and from thence onward atropine is to be instilled till the pupil remains of sufficient width.

Considerable complaint has been made of late because the operated eye is not examined for so long a time after the operation, but in reply we ask, *so long as our present therapeutics are of no avail if the section does not recover normally, why inspect the eye at all?*

In reflecting upon the demand that the patients ought to remain in bed three whole days, we must remember in cases of senile marasmus, asthma, emphysema of the lungs, etc., where the patient cannot lie so long recumbent, that the combined operation would better be performed.

RÉSUMÉ OF STATISTICS OF OPERATIONS AT PROF. SCHWEIGGER'S CLINIC.

1.—122 cases with capsular forceps gave:

Successes	89 =	72.9 %
Secondary cataracts	27 =	22.2 %
Losses	6 =	4.9 %
	122	100 %

2.—90 cases with puncture of the vitreous body gave:

Successes	57 =	63.4 %
Secondary cataract	25 =	27.8 %
Losses	8 =	8.8 %
	90	100 %

3.—159 cases with peripheral division of the capsule gave:

Successes	132 =	83 %
Secondary cataract	18 =	12 %
Losses	8 =	5 %
	159	100 %

Or, to sum up, 371 cases of extraction *with* iridectomy, with these results:

Successes	278 =	75.1 %
Secondary cataract	71 =	19.2 %
Losses	22 =	5.7 %
	371	100 %

Now our statistics of the *simple extraction* embrace 450 cases, resulting as follows:

Successes	346 =	76.5 %
Secondary cataract	84 =	19.1 %
Losses	20 =	4.4 %
	450	100 %

It was not our intention in this article to construct brilliant statistical tables (the terms success, secondary cataract, and loss, are flexible ideas), but to compare the results, in *the same clinic*, of the simple and the combined methods of cataract extraction. But the latter series of figures demonstrate that neither secondary cataract nor losses have increased since we have followed simple extraction of senile cataract. Q. E. D.

SUPPLEMENTARY REMARKS.

We have endeavored in the previous pages to exhibit the indications for the performance of iridectomy in the extraction of senile cataract, and any one can see that it is by no means our intention to make it "a principle to abandon the excision of the iris." On the contrary, there lies the essence of our remarks, that we are learning how to distinguish the cases which are better suited for simple extraction, and those in which the iridectomy is preferable. And that by further observations, as well as by the communications of other operators, we shall be enabled to decide with sufficient certainty when and how often to perform the simple extraction, so that we shall then be enabled to perform it with greater security and greater success. Therefore I deem it proper here to state in what proportion in this hospital the simple extraction stands to the combined.

When simple extraction was again resumed, we at first operated only upon those cases which seemed especially favorable; most were operated upon with an iridectomy. But later, encouraged by our excellent results, we operated on every normal case—that is to say, in the majority of cases, without the iridectomy. In other words, at various intervals between the performance of our last 400 simple extractions, we have performed 65 extractions with an iridectomy.

ON THE PROPOSED METHODS FOR NUMBERING PRISMS.

By A. DUANE, M.D., New York.

A.—MINIMUM DEVIATION.

THE proposition that prisms should be numbered according to their angle of minimum deviation instead of their refracting angle, while apparently satisfactory from a scientific point of view, is yet open to at least one objection on the score of scientific accuracy. This objection is that in practical ophthalmological work prisms are almost never placed in such a position as to produce minimum deviation. In the ordinary trial-frame, for example, a prism is placed with either its posterior or its anterior face perpendicular to the incident ray. The former (postero-normal) position is assumed when the prism is slipped into the front clip of the frame; the latter (antero-normal) position is assumed when the prism is placed in the back clip. The position of minimum deviation is never assumed. Moreover, even if one prism could be placed in the latter position, a series of superimposed prisms, such as are employed in testing or exercising the adductive power of the eye, could not be thus disposed. While these considerations invalidate to some extent the scientific advantages claimed for the proposed change, it remains to be seen whether the difference between the values of the deviation produced in the three different positions of the prism is sufficiently great to be of practical moment. In order to determine this, I have calculated the following

Rotary Prism

table representing the value of the deviating angle in these three positions. The index of refraction is assumed as 1.54.

Refracting-Angle of Prism.	Angle of Deviation in		
	Position of Minimum Deviation.	Postero-normal Position.	Antero-normal Position.
1°	0.5	0.5	0.5
2°	1.1	1.1	1.1
3°	1.6	1.6	1.6
4°	2.2	2.2	2.2
5°	2.7	2.7	2.7
6°	3.2	3.2	3.3
7°	3.8	3.8	3.8
8°	4.3	4.3	4.4
9°	4.9	4.9	5.0
10°	5.4	5.4	5.5
11°	6.0	6.0	6.1
12°	6.5	6.5	6.7
15°	8.2	8.2	8.5
20°	11.0	11.1	11.8
25°	13.9	14.0	15.6
30°	17.0	17.2	20.4
35°	20.1	22.1	27.1
40°	23.6	24.7	41.8

The facts to be deduced from this table are as follows:

1. Prismatic numeration according to the angle of minimum deviation is sufficiently accurate when a single prism is used, as we ordinarily employ no prism with refracting angle greater than 20°, and up to this limit the deviation produced is practically the the same in all positions.

2. When, however, as in testing the adductive capacity, an aggregation of prisms, whose collective refracting angle exceeds 20°, is employed, the resulting deviation will be more than the sum of the individual deviations produced by the component prisms, and must be computed by referring once more to the refracting angles of the latter. Thus, if two prisms having respectively deviating angles of 8° and of 11° are placed before one eye in the front clip of the trial frame, the resulting deviation will not be 19°, but, since the prisms have respectively refracting angles of 15° (nearly) and 20°, will be the deviation produced by a prism of 35° refracting-angle placed in the postero-normal position, or will be 22°.

3. When an object viewed through a converging prism arranged in the postero-normal position, is approached to the eye, the incident ray is no longer normal to the posterior face but becomes more and more nearly normal to the anterior face of the prism, *i. e.*, the prism is more nearly in the antero-normal position. Hence the deviation produced by the prism, although its position remains the same, is greater in proportion as the object approaches the eye; and when an aggregation of prisms of total refracting angle in excess of 20° is employed and the object is brought very near the eye, as in testing the absolute maximum of convergence [Maddox], the true convergence would be quite a little in excess of that calculated upon the assumption that the prism has remained in the postero-normal position, and still more in excess of the value deduced from the minimum deviating power of the prism.

No system of prismatic numeration is altogether satisfactory, since, whatever system we follow, the number that we assign to any given prism holds good for but one position of it, and represents a value which is constantly varying with the variations in the relative places of the prism and the objects viewed through it. For prisms of very oblique refracting angles, it is true, this variation is slight and can be disregarded, and hence for all prisms which have a deviating angle of less than 10°, and especially for all prisms used in occulists' prescriptions, the new system is sufficiently accurate and, as it is preferable on other grounds, should doubtless be adopted. But in all experiments with abduction and adduction where we are constantly called upon to use prisms of higher power, the new system is not only scientifically inaccurate but is also inconvenient. For, as the example above given shows, it is not possible to determine directly from the individual deviating angles of a series of superimposed prisms the deviating angle of the series as a whole; while, if the prisms are numbered according to their refracting angles, the summation of a series of prisms is effected at once and without calculation. If, for example, we have superimposed in the trial frame a series of prisms, whose refracting angles taken all together amount to 40°, the deviating effect of such a series will be the same no matter how the prisms composing the series vary in strength and number. In other words, $\text{p. } 40^\circ = \text{p. } 15^\circ + \text{p. } 25^\circ = \text{p. } 1^\circ + \text{p. } 2^\circ + \text{p. } 3^\circ + \text{p. } 4^\circ + \text{p. } 5^\circ + \text{p. } 6^\circ + \text{p. } 7^\circ + \text{p. } 12^\circ$, and so on indefinitely. But, if the prisms were numbered according to their deviating angles, the first expression would become p. 23.6° (d), the second p. 8.2° (d), and p. 13.9° (d), the sum of which would be 22.1° (d); and the aggregate of the component deviating angles of the third expression would be 21.6° (d). And the actual deviation produced by such an aggregation (no matter of what prisms composed) would be 24.7°, if, as would almost always happen, it was placed in the postero-normal position. Hence, to determine the deviation produced by a series of prisms when the refracting angles are given, we have only to add together the refracting angles and find the deviation produced by a prism whose refracting angle is equal to this sum; but, if the prisms are numbered according to their deviating angles, we have to first ascertain the refracting angle of each and then perform the addition. Accordingly, in all problems involving the use of several prisms together, and particularly in testing and exercising adduction and abduction, the old system is much more convenient than the new; and it becomes a question how far the advantages arising from the introduction of the new system in the case of weak prisms would be counterbalanced by the disadvantages due to its use in the class of cases just described. In fact, it may well be asked whether it is worth while to supersede a system already well established by a new one which is not in all respects its superior either in accuracy or in convenience.

B.—METRE-ANGLES AND PRISM-DIOPTRIES.

If numeration according to the angle of minimum deviation is not perfectly satisfactory, the proposed systems of measurement by prism-dioptries and metre-angles are still less so. In the former system, in which prisms are numbered according to the tangent of their deviating angle, the additional complication is introduced that, as the tangents increase faster than their angles, the amount of deviation as measured by prism-dioptries is too great. 40 P. D., for example, denote an angle of deviation less than forty times that produced by 1 P. D.[1] Nor does there seem any advantage, but rather the reverse in substituting a linear for a circular equivalent of the value of an angle.

As regards the metre-angle the case seems even worse. For, not only is the metre-angle a variable quantity, ranging between the limits of $1\frac{1}{2}^\circ$ and 2° according to the interpupillary distance, but further, with the higher multiples of the metre-angle, the angle of convergence is not exactly proportional to the nearness of the point of convergence to the eyes. The metre-angle, for instance, for an inter-pupillary distance of 60 *mm* is 1° 43′—*i. e.*, this is half the angle of convergence of the optic axes at the distance of one meter. But 15 m.-a. representing a convergence of the optic axes of $51\frac{1}{2}^\circ$, corresponds to a point of convergence of the optic axes, situated not at 66.7 *mm* ($=\frac{1}{15}$ metre) but at 62.1 *mm.* Nor is this all. This lack of correspondence between the angle of convergence and the distance from the eye of the point of intersection of the converging optical axes, is itself irregular, being different for each inter-pupillary distance. Thus for an inter-pupillary distance of 60 *mm*, 15 m.-a. correspond to a distance of 62.1 *mm* of the point of convergence; for an inter-pupillary distance of 70 *mm*, they correspond to a distance from the eye of 60.5 *mm.* The expression 15 m.-a., therefore, means not only a degree of convergence varying with each eye, but also means a point of convergence situated at varying distances from the eye. To number prisms according to a value so indeterminate seems surely most unwise.

NOTE.—Although not strictly belonging to this subject, it may not be amiss to note here an error which, doubtless through inadvertence, has crept into Maddox's book on "Prisms" and which has been copied in Prentice's article on "The Prism-Dioptry" (ARCH. OF OPHTHALM., xix., 2 and 3, p. 128). In treating of the composition and resolution of prisms, Maddox gives the formula:

$$(\text{I.})\ \text{Sin.}\ x = \text{Sin.}\ \Delta\ \text{Sin.}\ r,$$

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[1] The exact values are: 1 P. D. = 34′ 22.8″, hence 40 × 1 P. D. = 22° 55′; but 40 P. D. = angle corresponding to a deflection of 40 cent. at 1 metre = 21° 48′.

where x is the lateral displacement produced by a prism of deviating angle Δ, which is rotated through an angle r from the vertical. Further, for the angle of deviation R of the prism representing the resultant of a vertical prism of deviating angle V and a horizontal prism of deviating angle H, he gives the formulæ:

$$(2.)\quad \text{Sin. } R = \sqrt{\text{Sin.}^2\, V + \text{Sin.}^2\, H},$$

and

$$(3.)\quad \text{Sin. } r = \frac{\text{Sin. } H}{\text{Sin. } R}.$$

Here r is again the angle which the axis of the resultant prism makes with the vertical. Here a little consideration of the trigonometrical principles involved will show that these formulæ should read as follows:

$$(1.)\quad \text{Tan. } x = \text{Tan. } \Delta \text{ Sin. } r.$$

$$(2.)\quad \text{Tan. } R = \sqrt{\text{Tan.}^2\, H + \text{Tan.}^2\, V}.$$

and

$$(3.)\quad \text{Sin. } r = \frac{\text{Tan. } H}{\text{Tan. } R}.$$

This error, however, is of no practical importance, since for angles, such as are here under consideration (viz., those less than 10°), the sine, tangent, and arc may without sensible error be taken as identical. Indeed, these formulæ may as well be written (by substituting arc for tangent) in the simpler forms:

$$(1.)\quad x = \Delta \text{ Sin. } r, \text{ and } y = \Delta \text{ Cos. } r,$$

(where y represents the vertical displacement produced by the inclined prism, just as x represents the lateral displacement).

$$(2.)\quad R = \sqrt{V^2 + H^2}.$$

$$(3.)\quad \text{Sin. } r = \frac{H}{R}.$$

By the aid of the first of these simplified formulæ, I have been for some time in the habit of calculating the respective horizontal and vertical deviations produced by a revolving prism employed for testing vertical and lateral insufficiencies.

TEMPORARY EXOPHTHALMOS AND OPHTHALMOPLEGIA EXTERNA FOLLOWING GRIP.[1]

BY PETER A. CALLAN, M.D.

Bernard F. B., aged 44, native of United States, came to my clinic at the N. Y. Eye and Ear Infirmary, January 10, 1890, on account of the extreme protrusion of both his eyes. Until within the last three weeks his health had been good, and most of his life had been spent at sea, as steward in the service of the Pacific Mail S. S. Co. During the past year he was storekeeper for the Phœnix Construction Company. He was a man of intemperate habits, and of a very nervous, excitable disposition.

He had an attack of the grip beginning Christmas, 1889; but, with the exception of two days, this did not prevent his attending to his regular duties as storekeeper. The attack left him very weak, and harassed by an annoying cough. January 7, 1890, two weeks from the onset of the grip, he began to suffer from a most unbearable headache, which having lasted twelve hours, the right eye "began to bulge out of its socket" (to quote his words), and in about twenty-four hours the left commenced to similarly protrude. For about thirty-six hours his suffering from his head and cough became so severe that friends watched him closely fearing he might commit suicide.

When both eyes became exophthalmic, his headache diminished very much in severity; and when admitted as a patient, January 10th, had almost disappeared.

Patient was fair, about medium height, and full habit, weighing, I should judge, about one hundred and eighty pounds. Temperature, when admitted at 3 P.M., was 102°; pulse, 120. Owing to his condition, vision not taken. Both eyes were extremely prominent and immobile, with chemosis below, reaching almost the level of lower eyelids. Pupils moderately dilated.

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[1] Read before the Ophthalmic Section of the N. Y. Academy of Medicine, February, 1890.

Ophthalmoscope showed nothing characteristic. Slight blur of optic nerve outline, with tortuous veins on each retina. Patient was put to bed, a saline cathartic given, and leeches applied to his temples ; 10 P.M. of same day, temperature 104°, mild delirium. For twenty-four hours after admission there was not any marked change in the general condition of the patient, except with regard to the exophthalmos. The saline cathartic operated very freely in due time, and apparently as a result there was marked diminution in the amount of eye protrusion. The eyelids could be readily shut, which was impossible on the day of admission, and the eyeballs could be moved to a considerable extent. Patient's condition grew worse, and on second day, temp., 105° ; pulse, 130 ; pupils became contracted ; delirium more pronounced ; and he sank into a comatose state, and died January 13th, at 9 A.M., as if from apoplexy.

A few hours prior to death, the exophthalmos practically disappeared, but the chemosis remained in part. At 2.30 P.M. of same day, **autopsy** by Drs. Harvey and Gibson, of the house staff. On removing the calvarium, the meninges were found adherent to the cerebrum, and very dark in color. Between the dura and pia mater, covering the optic groove, olivary process, sella turcica, and extending laterally over both sphenoidal fissures, there was found a circumscribed sac of serous fluid holding between three and four drachms of serous fluid. The optic nerves in their passage into the orbits were found flattened in the foramina, and fluid escaped from orbits. In the lateral ventricles, especially the right, there was found considerable serous fluid. In removing the brain, an unusual amount of fluid escaped.

The case is unique, on account of the transitory character of the exophthalmos and the cause. The patient had originally a strong physical constitution, which the abuse of alcohol had impaired. The harassing cough which resulted from the grip tended to keep the brain capillaries congested, and, owing to impoverished condition of the blood, diapedesis took place—serous effusion. The most extraordinary feature was the sacculated bag of fluid which tightly compressed the sphenoidal fissures and optic foramina, causing ophthalmoplegia externa and exophthalmos of both eyes, both conditions disappearing before patient's death.

A CASE OF DISSEMINATED SCLEROSIS, PRESENTING THE CLINICAL ASPECT OF PRIMARY SPASTIC PARAPLEGIA, WITH ATROPHY OF BOTH OPTIC NERVES.

By Dr. CHARLES ZIMMERMANN, Milwaukee, Wis.

THE frequency of ocular symptoms in disseminated (insular) sclerosis is a well-established fact, and, from the observations of Charcot, Erb, Leyden, and Pitres, we know that primary spastic paraplegia may be the incipient stage of disseminated sclerosis, but, in regard to the occurrence of affections of the ocular nerves, especially the optic nerve, in primary spastic paraplegia and their diagnostic value, literature gives very little information. Erb, in his first publication on primary spastic paraplegia (*Berliner klinische Wochenschrift*, 1875, p. 358), says: " No cerebral nerves and no cerebral functions are altered in any way." Gnauck (On Eye-Affections in Disseminated Sclerosis, *Berl. klin. Wochenschrift*, 1884, p. 421) mentions five cases of primary spastic paraplegia, in which ocular disturbances never occurred. Gowers, 1886 (" Primary Spastic Paraplegia " in " Diseases of the Nervous System," vol. i., p. 333): " Ocular symptoms are extremely rare. The action of the pupils is usually perfect, and I have only once seen optic-nerve atrophy in an uncomplicated case." In the third edition of his " Medical Ophthalmoscopy," of 1890, he says, under the heading, " Lateral Sclerosis ": " Ophthalmoscopic changes are very rare in cases which present the symptoms of primary lateral sclerosis of the cord. In one or two cases, however, I have seen gray atrophy slowly supervene, similar in character to that met with in locomotor ataxy."

Peltesohn (*Centralblatt für prakt. Augenheilkunde*, 1886, p. 106): As great a rarity is furnished by a "case of primary spastic paraplegia with atrophy of both optic nerves in a syphilitic patient." "In my whole literary search I find only one analogous case, but only apparently: Suckling (*Brit. Med. Jour.*, 1882, p. 769) publishes a case of lateral sclerosis in a man of twenty-seven years of age, suffering from optic neuritis in one eye, iritis in the other, of syphilitic origin. An uncomplicated simple atrophy of the optic nerve in lateral sclerosis did not come under my observation." Vossius (*Grundriss der Augenheilkunde*, 1888, p. 363: "Uncomplicated atrophy of the optic nerve is very much rarer in spastic tabes than in locomotor ataxy." Michel ("Lehrbuch der Augenheilkunde," ii. Aufl., 1890, p. 518) mentions the occurrence of atrophy of the optic nerve in primary spastic paraplegia, and that its propagation on anatomical paths was supposed (through the corpora restiformia, the lower portion of the pons, the pes, the white substance, the thalami optici, and the capsula interna to the tracts). Oppenheim ("Zur Pathologie der disseminirten Sclerose," *Berl. klin. Woch.*, 1887, p. 904) was the first to devote a special investigation to this subject, giving it its due importance. He found in 11, out of 20 cases of primary spastic paraplegia of his own observation (*i. e.*, in more than 50 per cent.) a pathological condition of the optic nerve, and in the post-mortem examinations of 5 cases sclerotic foci in the optic nerve, chiasma, and optic tract. He calls attention to the fact that *disseminated sclerosis presents much more frequently than is generally supposed the clinical features of primary spastic paraplegia*, and that *one symptom*, which has to be looked for, the *affection of the optic nerve* ("described with special precision and completeness by Uhthoff and Gnauck"), *may insure the diagnosis*. He asserts that primary spastic paraplegia, with simultaneous affection of the optic nerve is not an unfrequent clinical form of disseminated sclerosis.

Recognizing the importance of Oppenheim's observations, and considering the scarcity of publications on this subject (Bruns reports a similar case, *Berl. klin. Woch.*, 1888, p. 90), I think it will be of sufficient interest to describe the following case, in which the atrophy of both optic nerves, with the predominating clinical appearance of primary spastic paraplegia, guided me to the diagnosis of disseminated sclerosis.

W. F., painter, twenty-six years old, came under my observation on November 27, 1886. He complained of dimness of sight in right eye for about eight weeks and of diplopia. V, R $= \frac{15}{70}$; V, L = 1. Field of vision and perception of colors normal. Both pupils equal, reacting perfectly normal upon light. The ophthalmoscopic examination revealed the *temporal two thirds of both optic discs perfectly white*, all the vessels being crowded to the normally colored nasal third. He had *crossed diplopia*, commencing on the median line and more noticeably on the left side, the distance of the double images becoming greater when looking to the left, from a *paresis of right internal rectus.* Under the use of iodide of potassium and subcutaneous injections of nitrate of strychnia his sight improved, so that when I saw him last, on January 31, 1887, V in both eyes was = 1, and diplopia had disappeared. *But the ophthalmoscope showed the same atrophic condition of the temporal two thirds of both optic discs*, as at the first examination. He resumed his business as a painter, and a few months later, through some of his friends, I heard that he was not troubled any more with his eyes.

On March 9, 1891, he came again to my office, sent by his family physician. He did not complain about his eyes, but I at once noticed from his peculiar gait that he must have an affection of his nervous system, remembering at the same time the atrophy of his optic nerves. Upon closer examination I found all the *symptoms of primary spastic paraplegia*, with some complications, which led me to the diagnosis of *disseminated sclerosis.* But, before I go into the description of the case, I give the previous history of the same, which may throw some light on the etiology, as well as it may corroborate the assertion of some authors (Oppenheim, Bruns), that the causes and the incipient stages of primary spastic paraplegia and of disseminated sclerosis may be traced as far back as the earliest childhood.

History: Both parents of the patient and two brothers are living and healthy. There is no inherited neuropathic tendency in his family. Except measles no acute infectious disease. According to his mother's statement, he has been stammering since his fifth year, getting worse when excited. In school it was sometimes so bad that he hardly could read. When he wanted to pronounce a word commencing with the letter "d" he could not get it, unless he began reciting the alphabet and uttering "a, b, c," then he could say "d." At the age of eight years he fell from a tree on his back without apparent injury, and when nineteen years old he suffered a severe exposure to cold by breaking through the ice while hunting, being wet all over for several hours. About that time he noticed sometimes a certain awkwardness in his movements—for instance, when in dancing-school he had danced a few dances as well to the right as to the left; he could not any more turn to the left, only to the right. In April, 1887 (soon after he had been under my treatment for his ocular affections), he had occasionally peculiar sensations of numbness and tingling in his right arm; it felt as if asleep. Then he lost the power in this arm, so that he hardly could lift anything for about seven months. Gradually, without any medication, his arm got better, and he thinks that it is as powerful now as it ever was. About a year or two later he gradually experienced the same symptoms at first in his right and then in his left leg. He felt a numbness and weakness in both, so that he got tired more readily than before; his legs felt heavy. Formerly he could walk twelve and sixteen miles, now he got tired when he walked about a mile. Now and then he had paroxysms of painful spasm in the muscles of the calf and the posterior muscles of the thigh. He had to rub his limbs in order to relieve the spasm. The left leg grew better, but not the right. After a further year (about a year ago) his arms, especially his right arm, which in the meantime had been well, again were invaded by the same progressive weakness and the peculiar sensory symptoms. Last January he commenced to feel weak around his abdomen and back, as if these parts were dead, but he had no pain in them. At the same time the sphincters became affected. He has no incontinentia, but a certain weakness, so that if he does not at once follow the calls of nature he is not able to resist them.

I examined the patient on March 9 and 10, 1891, and found the following condition:

Status Præsens.—The patient is a healthy-looking, tall, slender-built man (he says that he lost flesh—about fourteen pounds last winter). No exanthemata, no swellings of glands, no signs of lues. No intoxication with alcohol, tobacco, or lead. His muscles are not wasted, and there is no difference in the circumference of the corresponding limbs. His gait is characteristic of primary spastic paraplegia ; the right leg seems to drag behind the patient, and is hauled forward as a rigid whole, not being bent in the knee-joint, its great toe catching against the floor. (Gowers). He wears off his right shoe at the inside of the toe. Standing with closed eyes he has no tendency to fall over. On first rising in the morning he can walk very well, but after about fifteen minutes it gets worse, his legs feeling weaker then. In the flexor muscles of the hip in both legs, but especially in the right, there is a distinct loss of power, so that the thigh can only be lifted a very little height and cannot be brought into horizontal position while the patient is standing. When sitting, he cannot bring one leg over the other ; if he wishes to do this he must do it with his hands. The same weakness is observable in the flexors of the knee and the foot. Rotation of the foot cannot be effected. The tendon-reflexes are very much increased and excessive. The slightest tapping of the tendon of the quadriceps femoris causes several excessive and quick knee-jerks, and ankle-clonus is readily obtained. When the patient sits with the great toe resting on the ground, his leg commences to shake violently until he stretches it out. There is spasmodic stiffness in the leg, so that the knee can be passively flexed only by some force applied to it. He has paroxysms of trembling and spasm, which are brought about mostly by changes of temperature. When, after retiring, the bed gets warm he feels a tickling in his legs, which commence to shake and to tremble, then they become rigid and are extended ("clasp-knife rigidity"). At the same time he feels a painful cramp in his calf, which will be relieved when the extension of the limb is completed. His wife stated that she noticed twitching of his muscles while he was asleep (paroxysms of clonic spasm). Otherwise his sleep is good and not disturbed in the least. There is also a spastic rigidity in his abdominal muscles. When he bends the trunk to one side he cannot recover his position, but has to get hold of a table or his leg in order to raise his body. The right arm is affected in a similar way, but less in degree. There is weakness and diminished power in it and numb feeling in his fingers. On March 10th he felt for the first time contractions of the flexor muscles of his right forearm. The myotatic irritability becomes manifest by the excessive tendon reflexes on tapping the tendons of biceps, triceps, and flexor digitorum communis just below the wrist. The left arm is almost normal. No increased chin-reflex. Besides the subjective sensations of numbness and formication there is a defect of sensibility at the right forearm and hand. Commencing 2 *cm* above the olecranon the skin of the right arm is perfectly anæsthetic to the prick of a pin, extending over an area 7 *cm* long and 5 *cm* wide, starting from the internal condyle. The whole surface supplied by the ulnar nerve, except the dorsal portion of the fingers and the hand, shows impaired sensibility. He feels a stiffness in his fourth and fifth fingers and the corresponding volar portion of the hand ; it does not open and shut as it ought. Of the skin-reflexes the cremaster-reflex is diminished. He is unable to attend to his business as a painter, his right arm becoming tired of holding the brush, and the condition of his legs preventing him from climbing and standing on ladders and scaffoldings on account of the spasmodic rigidity of their muscles. His speech is hasty, one word overlapping the other when he is excited. It is not what has been termed " staccato or scanning utterance " (scandirende Sprache). Since January, 1887, no functional disturbances of the visual organ. No diplopia, no nystagmus. The action of the pupils is perfect. V in each eye = 1. He reads the whole day without inconvenience. No scotomata, visual field and color perception normal. But the temporal two thirds of both optic discs are just as white from atrophy as when first examined in November, 1886. The other senses normal.

In regard to mental symptoms (he is somewhat irritable) an undue contentment exists, which is distinctly unnatural, considering his inability to work for over a year. No headache, no vertigo. Memory good. Before falling asleep he frequently experiences visual hallucinations (he sees in the dark : clouds, gardens, frescoed rooms, horses, etc.). He has a very good appetite, no difficulty in swallowing, nor any other bulbar nerve-symptoms. Urine free from albumen and sugar. His sexual power is remaining (he is married and has a child six months old).

The chief symptoms of this case : weakness, myotatic irritability, spasms and their gradual onset, constitute the clinical features of *primary spastic paraplegia* (primary lateral sclerosis). *Why then is this a case of disseminated sclerosis*, which I asserted it was? The *reasons* which led me to this diagnosis will at once become apparent when we *analyze* the *complications* which *our clinical history* contains. The most important of all is the *partial atrophy of both optic nerves;* the development and the appearance of atrophy in this case are characteristic of disseminated sclerosis; the sudden onset of amblyopia (in one eye) and the speedy recovery of sight; the partial atrophy, affecting only the temporal two thirds, and its rapid development; the lasting ophthalmoscopic appearance of atrophy with amelioration of vision to its normal acuteness, and the discrepancy between the ophthalmoscopic condition and the perfect performance of the function of the optic nerves; its early appearance before any other symptom could be observed. Uhthoff found this in four cases ("Untersuchungen über die bei der multiplen Hirnsclerose vorkommenden Augenstörungen") and Gnauck in case "Hayman" (*Berl. klin. Woch.*, 1884, p. 421). It was only about eight weeks before my first examination that the patient complained of failure of sight, and then his optic nerves were atrophic and have remained so ever since, the vision returning to its normal condition in about one month. An explanation of this peculiar course of atrophy of the optic nerve was first given by Uhthoff (*Berl. klin. Wochenschr.*, 1885, p. 245), who proved, from three cases of his own and four cases of Gnauck (*l. c.*)—*i. e.*, about 25 per cent. of the cases of disseminated sclerosis with ophthalmoscopic changes, that the *atrophy was of neuritic origin.* In disseminated sclerosis the optic neuritis turns very rapidly into atrophy, so that the patients very often come under our observation when the pathological changes of the optic nerve have already taken place, and then it is usually impossible to recognize the neuritic origin from the condition they present. Ch. Achard and Louis Gunion (*Arch. de Méd. Expérim. et d'Anat. Pathol.*, 1889, No. 5; abstract in *Brain*, part iii., 1890, p. 430), in a paper on "acute disseminated myelitis with double optic neuritis," described a case in which "spastic symptoms developed and sight was restored to a certain extent from blindness, which had come on in the course of six days. The patient died of acute phthisis. On subsequent examination three large patches of sclerotic change were found—one in the upper cervical region of the cord, another in the dorsal region, and a third in the optic nerves and tracts. In connection with these, secondary degeneration had taken place. Achard and Gunion, however, do not venture to come to any conclusion as to the nature of their case."

Very likely our patient had at first optic neuritis, which may have changed very early into atrophy in the period of eight weeks, or even less, so that when he came to the first examination the inflammation had subsided for some time, and the improvement of sight had already commenced. Uhthoff found as the cause of this retrobulbar neuritis sclerotic changes in the optic nerve (Untersuchungen, etc.). The medullary sheath of the nerve fibres becomes atrophic from proliferation of connective tissue in the septa, but the *axis-cylinders mostly remain unimpaired.* This is evidently the reason that the sight may return to its normal state, and that *very often no visual disturbances are complained of, where only the ophthalmoscopic examination reveals the atrophy.* Cases of this kind are mentioned by Oppenheim (*l. c.*). That the *atrophy* is confined to the *temporal two thirds*, as in our case, or the temporal halves, is *quite common* (Charcot, Gnauck, Uhthoff, Oppenheim), and even if the nasal half becomes also affected, the temporal portion shows the changes more markedly (Gnauck, *l. c.*).

According to Oppenheim, the atrophy of the optic nerves, with the symptoms of primary spastic paraplegia, would be sufficient to justify the diagnosis of disseminated sclerosis. But in order to complete our investigation we have to consider some other symptoms, presented by our case, which will help to support our diagnosis. The patient complained of *diplopia*, due to a *paresis of the right internal rectus* from an affection of a branch of the right oculomotor nerve. It seems most probable to assume that this paresis was caused by the development of a *sclerotic patch* at one of the *nuclei* of the third nerve, only affecting the branch which supplies the internal rectus as long as the incipient sclerotic process was irritating it by the inflammatory proliferation of connective-tissue elements of neuroglia. As soon as a shrinkage followed and the pressure ceased, which the inflammatory swelling had effected, the persistence of the axis-cylinders (characteristic of sclerosis) admitted of a perfect functional recovery. Affection of the ocular nerves with double vision is quite a frequent lasting or transitory symptom of disseminated sclerosis, and very often one of the first. Charcot observed disturbances of the ocular nerves as an inaugurating sign of the disease. Uhthoff (Untersuchungen, etc., *l. c.*) found paralysis of the ocular nerves, mostly abducens and oculomotor nerve (the former twice as much as the latter), in 17 per cent., *half of them* of decidedly *nuclear* character. In some of his cases the paresis was the *initial* symptom of the disease. He also (*Berl. klin. Wochensch. l. c.*) observed a case of *partial paralysis* of the left oculomotor nerve in a case where the levator palpebræ superioris and the superior rectus were affected. Bernhardt (Gehirnkrankheiten, " Real-Encyclopædie der gesammten Heilwissenschaft," von Eulenburg, ii. Aufl., Bd. viii., p. 99) says of the paresis of ocular nerves in disseminated sclerosis: " This affection is either central in the nuclear region, or a partial gray degeneration of single peripheral ocular nerves." But according to Mauthner (" Die Nuclearlaehmung der Augenmuskeln," p. 373, 1885), " the often slight ocular paralysis, preceding disseminated sclerosis is entirely a *nuclear* one, as we can now pronounce with certainty." Gowers (" Diseases of the Nervous System," vol. ii.) says: " In some cases there is increasing palsy of one of the ocular nerves, due to the development of sclerosis in the nerve trunk." The affection of the right oculomotor nerve in our case has all the remarkable clinical features observed in disseminated sclerosis; it is *partial, transient* and an *initial symptom.* Another complication deserves our attention: the *sensibility.* Oppenheim (*l. c.*) lays great stress upon its diagnostic value in disseminated sclerosis, and advises us to search for its disturbances in the course of our observation. Remak (" Eulenburg's Encylopædie," ii. Aufl., xvii. Bd., p. 475) considers alterations of sensibility in primary spastic paraplegia always indicative of some graver affection, *i. e.*, mostly disseminated sclerosis. Our case shows a form of *anæsthesia*, *confined to a small district* of the right forearm, which is most common in disseminated sclerosis according to Gowers' description, who says, "that the loss of sensibility is more often partial, involving only a small area of a limb, and it may or may not correspond to the motor palsy." Besides this limitation to a small area it has a *fugitive* character, and is changing in our case. Although it was sought for, it was not found until March 13, 1891, and on March 25th it had changed in its extent and degree. This *acute development* and its *transitory character* are *pathognomonic* of disseminated sclerosis, as pointed out by Oppenheim (*l. c.*), who claims that he has not seen this in any other disease. The analgesia in syringo-myelia may have some resemblance to it, as it may also occupy exclusively certain zones of skin and mucous surfaces (*vide* Blocq in "*Brain*," part iii., 1890). The *early* appearance of *weakness* and *myotatic irritability* in the *arms*, especially the right arm, would be rather a symptom of incipient disseminated sclerosis, than of primary spastic paraplegia, according to Remak (*l. c.*), although in the latter the arms suffer in some cases in a similar manner as the legs. The weakness of the *sphincters*, as in our case, is a symptom, observed in both diseases, disseminated sclerosis and primary spastic paraplegia, but its rather sudden onset, in a quasi-apoplectic form last January, seems to indicate disseminated sclerosis. It may last weeks and months, and then disappear for a time. Although the *articulation* of our patient is hasty and hurried, and not of the well-known scanning character, we have to mention it as perhaps connected with the disease in view, since Bernhardt (*l. c.*) describes a case which showed the same hasty and precipitate *speech* at the very beginning of disseminated sclerosis, and Oppenheim (*l. c.*) observed cases in which patients, who exhibited the symptoms of disseminated sclerosis in the second and third decennia having developed them in the last years, recollected that their speech had been affected *since infancy.* In our case it was noticed in the patient's fifth year. Our patient's *undue contentment* with his *unfortunate condition* and the irritability of his disposition may be added here as further signs common in disseminated sclerosis.

Reviewing once more the complications of the clinical features of primary spastic paraplegia, exhibited by our case, they all seem of inferior importance in comparison with the affection of the *optic nerves*, which was the first, the only lasting, and the most objective of all. We learn from our case, *that atrophy of the optic nerve, when associated with the symptoms of primary spastic paraplegia, is the most valuable and reliable guide in the diagnosis of disseminated sclerosis.* When observed early, before any striking appearance of other symptoms, its peculiarities (set forth above) will give us a hint to be on the lookout for other signs, even in their most primary stages, thus enabling us to make an early and distinct diagnosis.

PUNCTURED WOUNDS OF THE EYE IN CHILDREN.

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IT is frequently observed in the practice of medicine that diseases and accidents, especially of the more unusual forms, seem to run in streaks, as it were; and the following four cases of punctured wounds of the eye, all happening to children under ten years of age, and all occurring in a city of twenty thousand inhabitants, within the space of a month, help to bear out this observation.

The greater liability in children under the age of puberty to develop sympathetic ophthalmitis is well known; and in two of these cases, Nos. 1 and 3, this liability is illustrated. These four cases, while not numerous enough to become of value in themselves as statistics, show the marked tendency towards sympathetic troubles in children. The deformity resulting from the removal of an eye in young children, due to the lack of development in the side of the face from which the eye has been removed, as well as the disfigurement resulting from the loss of the eye itself, is to be mentioned to the parents of the children, but neither of these results should be considered when the sight of the child is in danger, as I judge it to be if an immediate enucleation is not done, unless the case can be under the direct charge of one who will watch for and place the proper interpretation on the slightest symptom of commencing trouble.

Case 1.—J. L. S., age three years, stuck the point of a pair of scissors into the right eye, about a week before I saw him. On examination a large punctured wound was seen in the inner and lower part of the cornea, extending into the ciliary region ; a large hernia of iris was entangled in this wound ; lens swollen and opaque, and filling the anterior chamber ; enucleation advised at once, but was declined. Directions were given to watch for any signs of irritation in the well eye. Ten days after this I was informed that the light was so painful to the eyes that the room was darkened all of the time. Also that the pain in the eyes and head prevented the child from sleeping much in the night. This condition had been present for several days. An examination showed the right eye in the same condition as when first seen, *the left eye much injected.* The right eye was removed at once ; solutions of atropine and cocaine were used in the eye, and mercurial ointment rubbed into the temples, but with no benefit ; the pain and irritation continued, and although the child was taken out of town, and the optic nerve resected, the sight gradually failed, and ultimately was lost.

Case 2.—Master B., age nine years, was struck in the left eye, the night before I saw him, by a piece of glass thrown by an older boy. On examination a small punctured wound was seen in the upper and inner border of the cornea, passing into the sclera ; a small piece of iris protruded from the wound. The protruding iris was clipped off, a solution of eserine was used in the eye, and strict orders given to watch for any signs of inflammation in both eyes. Four weeks have passed, and no signs of trouble have appeared in either eye.

Case 3.—W. J., age twenty-two months, fell and stuck a piece of stubble into the left eye, about a week ago. An examination showed a large wound extending transversely across the cornea, all of the corneal tissue opaque, so that no view of the deeper parts could be obtained ; T — 2 ; enucleation was proposed but declined. In about ten days the child was again brought to me with unmistakable signs of irritation in the well eye. The injured eye was removed at once, and in a few days all signs of trouble had vanished, and the child could play out-of-doors without the light troubling him.

CASE 4.—H. H., age four years, was struck in the right eye with an arrow, about an hour before I saw him. A large lacerated wound below the cornea, about one *cm* in extent and almost wholly in the ciliary region, was found ; iris and vitreous were protruding from the wound. The eye was removed on the next day, and the case has shown no signs of trouble since.

SUBJECTIVE LIGHT SENSATIONS FOLLOWING ENUCLEATION OF THE EYEBALL.

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THE following very interesting case came to the dispensary department of the Harrisburg Hospital in October, 1890, and was the first patient of the kind I ever had, and in the language of an eminent ophthalmologist, is "almost unheard of":

Miss —— P., aged thirty-three years, by profession a music-teacher; had *ophthalmia neonatorum* four days after she was born, resulting in almost total blindness in both eyes. Was entirely blind in L E, and in R E could only discern the sun, moon, and a few colors, until in 1880, when epileptic convulsions set in, having as many as forty to fifty per week. These finally ceased in 1888 under large and continued doses of potass. bromide.

In April, 1885, panophthalmitis began in R E, for which it was enucleated on May 2, 1885. Three days after enucleation she began to have subjective sensations of light, varying in color and degree of brightness, and which continued up to the time that I last saw her, which was in December, 1890. Her descriptions of these sensations of light were as follows.

It appeared as though there was a lighted candle in the orbit, most of the time of a bright white light; some days it resembled a red sunlight, and other days like twilight. The days resembling twilight she describes as being very pleasant, but as *they* were few in comparison to the others, their continued annoyance was very distressing, and she wished to be in total darkness if treatment would accomplish it.

The white light would occasionally become so bright as to cause intense nervousness. This nervousness would always diminish, and sometimes disappear after she had been lying down several hours and resting.

Occasionally she is in total darkness, but has had but two or three days of total darkness in five years. During the dark days, regardless of her overtaxing her nervous system, there is no increase in the light sensations, but when the light sensations are bright, excessive mental work compels her to quit work and lie down in order to get relief. During these severe attacks she suffers with a dull and heavy sensation through the temporal and occipital regions.

She would invariably obtain relief much quicker by lying on the left side. Going out into the fresh air would also afford her some relief. Cold applications to the orbit would do no good, but cold water forced in jets into the orbit would relieve her for five to ten minutes. She is less annoyed when she is not busily engaged in any work that requires considerable mental effort. Has two or three bright days in succession, followed by one not so bright.

As for permanent relief, all treatment seems of no avail. Large doses of potass. brom. and ergot, continued for some time, give her some relief. Two of my colleagues and myself examined the orbit very carefully, but could find no enlarged or bulbous condition of the orbital end of the optic nerve. Pressure within any part of the orbit had no effect upon the light sensations, nor caused any pain in any manner whatsoever. I will not at present attempt to give any physiological explanation of these phenomena, but will ask the following questions. Would a diseased condition of the orbital end of the optic nerve cause these symptoms, or would a hyperæmic or congested condition of the optic tract or visual centres, produce these phenomena? I will be pleased to hear personally, from any of the profession who have had similar cases.

TREATMENT OF GRANULAR EYELIDS BY CANTHARIDATE OF POTASH.

By J. SANTOS FERNANDEZ, M.D., Havana, Cuba.

I DO not pretend to write extensively upon the application of cantharidate of potash in this most troublesome disease, which has put to hard work the mind of the ophthalmologist of all times. To-day I shall only point out the favorable changes which have been effected in those patients under my care submitted to the above treatment, reserving for a more complete article the question of whether we may accept or reject the results, which as yet can only be considered as reasonable hopes.

As granular eyelids are frequently observed in every ophthalmic dispensary, although not so much in Cuba as in other countries where poverty is more prevailing, every specialist has been able to try for this disease such remedies as would seem to promise real benefit.

Not long ago the use of jequirity was claimed by many as positively successful. This new substance I submitted to a careful test, the results of which I reported in several papers which I read before our Royal Academy of Medicine and Natural Sciences. Although this mode of treatment is undoubtedly beneficial to a certain extent, it is also true that it is not entirely free from danger, as proved in some of my cases. The same is my opinion as to the use of gonorrhœal pus, particularly recommended in Belgium; in this case the *therapeutic disease* is so severe and so important the organ to which it is applied, that I have always proceeded with the utmost care, considering the particular conditions under which our perpetual summer places the patients. I use jequirity sometimes anyhow, for every treatment has its opportunity, and its clinical value is determined by the right selection of the cases.

If the granular eyelids have been energetically treated by medical resources, the surgical methods suggested for them have been still more severe. One of the methods proposed is Galezowski's, which consists in the excision of the *cul-de-sac*, and which has met with strong opposition among his adversaries. This method was submitted by me to a most careful examination, and was the subject of several papers. It has been said that the excision was always followed by serious accidents, such as the exaggerated retraction of the surrounding tissues of the eyelid. My experience has not confirmed this assertion, some of my patients having been under observation fifteen years after the operation.

After this it will be easily considered that I was well prepared to try the cantharidate of potash injections as soon as I learned that it could improve to some extent the condition of such patients as had been subjected to a useless treatment during a long time.

Dr. Oscar Liebreich's communication to the Med. Society of Berlin (session of February 25, 1891) suggested to me the idea of trying this method. Liebreich considers the particular action of the cantharidine as due to the fact, well established by Buchner, that blood serum enjoys bactericidal properties; the cantharidine injections producing an increased flow of serum, especially in those organs where an inflammatory process takes place, it results that the method can be extremely favorable as one of the so-called elective remedies.

One of the patients treated by Dr. Liebreich for a laryngitis tuberculosa was under the care of Dr. Gutmann for a double trachoma since 1887, and had been treated all the time with sulphate of copper. Fourteen days after Liebreich's treatment was pursued, it was observed that the left eye was little irritable, and its conjunctiva smooth, and in the right eye the conjunctiva still slightly thickened and beset with very few granulations, which were removed for microscopical examination. Dr. Gutmann acknowledges that one single case proves little, but thinks that the prompt healing of the conjunctiva in this case is a fact deserving further studies.

I have tried, by common consent with my friend, Dr. Diago, the cantharidate of potash in five patients, from the 1st to the 20th of April, 1891; at this moment being obliged to leave for Europe, Drs. Madan and Acosta will carry on the observations, in order that I may on my return make an exhaustive report.

CASE 1.—J. A., Chinese (No. 23,021), æt. forty-three; has been sick since September 25, 1887. Actual cautery was applied to left eye on account of the existence of thickset granules; after that he has been treated with sulphate of copper, cadmium, and silver iodide; during this treatment no increase was observed as to the granulations. First injection of 0.05 *mgm*, which dose was increased to 0.1 *mgm.* The eighth day some uneasiness appeared, which prevented him from coming to my office. To-day the conjunctivæ are more pale; no positive improvement can be detected in this case, which will continue under the same treatment.

CASE 2.—A. B., yellow girl (No. 21,971), æt. fifteen. The diagnosis, "granulations and *pannus granulosus*," was established in December, 1889, but regular treatment was not pursued until September 25, 1890; this treatment consisted in frequent cauterizations, which succeeded in removing the acute state of the disease. When the injections were begun, the irritability was not very great. After a treatment of twelve days the conjunctivæ were smoother, but the patient, being exceedingly timid, refused more injections.

CASE 3.—J. R. G. (No. 25,077), æt. thirty-two, was first examined on March 3, 1891. Diagnosis: acute granulations, with suppurative keratitis in both eyes. The injections were first applied in the temples, and later in the arms. But the temples being painful, and the patient afraid, further injections were adjourned.

CASE 4.—M. V. y C., a girl æt. eleven; granulations since July 5, 1888; treated with ordinary remedies. On second day, first injection to right temple; third day, second injection to left temple, and afterwards in the arms. After ten injections, less injection of the conjunctivæ was noticed, and better appearance. I cannot as yet, nevertheless, affirm any marked change. Treatment will be carried on.

CASE 5.—M. C. (No. 4,847). This patient was treated long ago for an opacity of both corneas. Has suffered many acute attacks. The injections have been applied every other day ; the condition of the conjunctiva has greatly improved, it being less injected and smoother. The patient himself has noticed the advantages of the method, and is perfectly disposed to continue.

Although I have observed more cases than Dr. Gutmann, I cannot as yet assert anything positive ; I publish these few cases because they prove that if this method of treatment is not really beneficial it is at least harmless.

It was certainly a mere coincidence that called Dr. Gutmann's attention to this treatment, but after all, I think its use in granular eyelids is supported by good reason because of the analogy between granulations and tubercle, the tubercle consisting chiefly in cells which contain the specific bacillus ; these cells later undergo fatty degeneration. The lymphoid cells of the granulations disappear, leaving behind them connective tissue, which is also apt to degenerate.

Fatty degeneration of the granulations has been observed, although very seldom. The tubercle when degenerated may act as a foreign body with infectious properties, and hence give rise to suppuration and ulcers ; the granulations disappear like the cutaneous gummas, and often leave behind them a scar without previous suppuration.

HAVANA, April 24, 1891.

REPORT OF TWO HUNDRED CATARACT EXTRACTIONS.

BY DR. ROSA KERSCHBAUMER, OF SALZBURG.

Translated by Dr. CASEY A. WOOD, of Chicago.

THIS report includes two hundred cases operated upon between May, 1881, and January, 1883, and is a continuation of two previous hospital reports covering the time between 1878 and 1880 inclusive.

The total number of operations performed since the publication of the above mentioned communications to the end of 1888 exceeds five hundred. Of the remaining three hundred, as well as of others undertaken since, I shall publish a further report.

I.—METHOD OF OPERATION.

All the extractions were performed by the modified Graefe method, including a conjunctival flap and a small iridectomy. Great importance was attached to the careful and proper reposition of the edges of the cut iris. Except when the capsule was thickened it was opened by the cystotome; in the former case the capsule forceps were used. The lens was then delivered by the pressure of a Daviel's spoon on the cornea, cortical remains being removed by massage with the lower lid.

Asepsis was sought to be established both during and after the operation.

The instruments were first disinfected in alcohol and then in a solution of carbolic acid. For the past four years we have been in the habit of placing them, just before using, in boiling water.

In earlier years the dressing was carbolized cotton-wool. Since then we have used absorbent (Bruns) cotton, which has been soaked in a solution (1:5000) of sublimate. This solution is also employed for cleansing the hands, the conjunctival sac, and the parts about the eye.

II.—ACCIDENTS ATTENDING THE OPERATION.

A.—ESCAPE OF VITREOUS.

This happened thirteen times; eight times with uncomplicated and five times with complicated cataract. In no instance did loss of the eye occur. The following is a brief account of each case.

a.—Simple Senile Cataract.

J. E., æt. seventy-eight. Had senile marasmus. Cataract (R.) mature. Excision of iris followed by considerable loss of vitreous in consequence of patient's "screwing up" his eye. Chronic cyclitis. At his own request patient was discharged with cyclitis and V of $\frac{6}{60}$. Five months later returned with occluded pupil. After iridectomy sight could be improved from $\frac{1}{\infty}$ to $\frac{6}{24}$. Vitreous opacities.

R. L., sixty years old, during removal of lens (R. for mature cataract) forcibly contracted orbicularis. Normal recovery. V $\frac{6}{12}$.

J. V., sixty-nine years. Senile marasmus. R. mature cataract. During iridectomy on L. (hypermature cataract) nucleus receded and hyaloid presented in the wound. There was a moderate escape of vitreous, the lens being extracted by the aid of Weber's scoop. Good recovery. V $\frac{6}{20}$; later $\frac{6}{24}$. Cloudy vitreous.

W. G., æt. sixty-four. Hypermature cataract. Nucleus sank after excision of iris. Lens removed in its capsule. Loss of vitreous. Normal recovery. V $\frac{6}{18}$; subsequently $\frac{6}{12}$.

L. J., æt. sixty-three. L. mature cataract. Patient pressed out quite a quantity of vitreous. Could not remove cortical substance. All went well until eighth day, after which slight cyclitis. V $\frac{6}{60}$. Secondary cataract.

K. H., æt. seventy-four. L. hypermature cataract. During removal of cortical remains a moderate amount of fluid vitreous escaped. Thickened capsule remained. Recovery normal. V $\frac{6}{36}$. Vitreous opacities.

W. J., æt. sixty-three. Morgagnian cataract R. Semifluid vitreous escaped after corneal incision. Extraction by means of Weber's scoop. Fluid cortex. Cataract spherical. Good recovery. V $\frac{6}{9}$.

S. G., æt. sixty-two. R. hypermature cataract. A small amount of fluid vitreous lost during removal of cortical remains. Recovery normal. V $\frac{6}{9}$; later $\frac{6}{6}$.

b.—Complicated Senile Cataract.

Th. H., æt. sixty-nine. L. hypermature cataract. Obliged to enlarge primary incision. Nucleus extracted with difficulty. Escape of vitreous. Slight cyclitis during recovery. Hazy vitreous; choroiditis. V $\frac{6}{36}$.

B. Th., æt. fifty-eight. Hypermature cataract. Iridodonesis. After completing incision vitreous presented. Lens with capsule delivered by means of the scoop. Moderate loss of vitreous. Normal recovery. V $\frac{6}{36}$. Vitreous opacities.

W. A., æt. sixty-four. Adherent cataract L. During removal (July 7, '82) a large amount of vitreous was lost. Normal recovery. V $\frac{6}{36}$. Vitreous opacities.

M. M., æt. sixty-seven. Complicated hypermature cataract L. During removal of cortical remains a bead of vitreous escaped. Recovery normal. V $\frac{6}{12}$.

F. C., æt. thirty-seven. Mature cataract. Excessive myopia; choroiditis, and vitreous opacities. Patient, otherwise healthy, says she has had weak and short sight since early childhood. Extraction Jan. 10, '82. Corneal incision followed by escape of fluid vitreous. Cataract and entire capsule delivered with scoop. Good recovery. V $\frac{6}{24}$.

S. E., æt. fifty. R. traumatic cataract. Iridodonesis. After incision vitreous. Cataract in capsule extracted by means of Graefe scoop. Considerable loss of vitreous. V $\frac{6}{36}$. Vitreous opacities.

B.—EXCESSIVE HEMORRHAGES.

These occur (1) from the conjunctiva, (2) after iridodialysis, and (3) spontaneously. The last is the result of senile changes only.

In every one of this series of cases in which the accident occurred the blood was absorbed without producing inflammatory symptoms and without noticeable effect upon either the convalescence or the visual acuity.

III.—AFTER-TREATMENT.

In cataract extraction the after-treatment plays an important part, not only as regards the medical attendance and the nursing, but the condition of the patient must also receive consideration. The greatest care should be observed in applying and renewing the antiseptic dressing. At first we used dry carbolized cotton-wool and flannel bandage, but this was later on discarded for sublimated absorbent cotton with which all patients are now dressed during the first week. In addition to this there is placed at the inner canthus between the puncta lacrymalia a small sublimate tampon, sprinkled with iodoform. By this means all secretions from the canaliculi are carried off through the interpalpebral aperture, or are, at least, disinfected. I have found this little plug to possess another advantage: it drains off the tears which would otherwise collect in the conjunctival sac and annoy the patient.

Following the example of my teacher, Prof. Arlt, I allow those patients in whom the operation was without accident to sit up in an arm-chair forty-eight hours after extraction. I find that, as a rule, they are quieter under this treatment and sleep more soundly at night.

IV.—ACCIDENTS OF CONVALESCENCE.

a.—Primary Suppuration.

I can recall but one example of primary purulent infection of the wound among all the cases included in this report.

R. M., æt. seventy-one. L. hypermature cataract. R. phthisis bulbi. Patient is a sad example of our mountain cretinism. She had, two years before, been operated upon elsewhere for cataract in right eye. After an entirely satisfactory extraction (Aug. 18, '82) patient repeatedly tore off the bandage. Wound became infected in twenty-four hours, then purulent iridocyclitis, and finally panophthalmitis.

b.—Cyclitis.

This occurred in twelve cases. In seven there was incarceration of the iris, in three the cataract was complicated, and in two the patient was marasmic.

St. M., æt. seventy-four. Cyclitis. When discharged V $\frac{6}{36}$; in about two months $\frac{2}{60}$; cyclitis at an end; dense secondary cataract.

E. J., æt. fifty-five. Cyclitis on tenth day. Vitreous opacities. When discharged V $\frac{6}{60}$, which improved spontaneously until in six months it was $\frac{6}{36}$.

H. U., æt. sixty-five. Discharged in good condition on tenth day. V $\frac{6}{36}$, with secondary cataract. Used his eyes too soon and imprudently, and in consequence the eye operated upon became painful, red, and tender. After eight weeks he returned to me suffering from severe iridocyclitis and occluded pupil. Contrary to my advice, he would not remain in the hospital but left for home. Two years afterwards I learned that he had died *blind in both eyes.*

E. A., æt. sixty-eight. Nearly mature cataract extracted April 21, '82. Cyclitis. V $\frac{6}{24}$.

S. A., æt. seventy-five. Small wound; large nucleus; delivery difficult. Iris incarcerated at both angles; occlusion of pupil. V$\frac{1}{\infty}$.

B. J., æt. sixty-two. Incipient cataract R. Reopening of wound on second day. Incarceration of iris in external angle. Cyclitis. V $\frac{6}{60}$.

T. T., æt. sixty-three. R. extraction. Iris in outer angle. Eighth day after operation severe cyclitis. On the twenty-sixth day V $\frac{1}{60}$. A year later I saw him again in consultation, and found an occluded pupil in the right eye and evidences of a recent *sympathetic* affection of the left. Doubtful light perception in both eyes.

S. E., æt. seventy-seven. Marked collapse of cornea, with cyclitis and keratitis striata. V $\frac{6}{18}$.

c.—Incarceration of Iris.

This resulted in thirteen cases; six of these recovered perfectly, but the remaining seven had cyclitis, as described above.

As a rule, incarceration occurs when in consequence of an inaccurate iridectomy or imperfect reposition of "tags" of iris, the latter becomes entangled between the lips of the incision. This accident also happens, though less frequently, if the wound should be ruptured; the aqueous escapes and draws with it into the wound not only cortical remains but the sides of the coloboma—however perfect the position of the latter may have been.

d.—Keratitis Striata.

This was present in 6–8 % of the operations, and almost invariably in patients suffering from senile marasmus. With one exception—complicated by cyclitis, the result of an imprisoned iris—the cases were unimportant.

e.—Rupture of the Corneal Wound.

There were five of these cases; two had in consequence corneal astigmatism, while the other three ran a normal course.

In my opinion this accident is not a serious one so far as the safety of the eye is concerned, unless infection of the wound sets in, or incarceration of the iris occurs, or unless it is the result of considerable violence. In the last instance we may have loss of vitreous, extensive intraocular hemorrhage, or even retinal detachment. On the other hand, the visual acuteness is very likely to suffer, especially when the injury happens several days after the extraction. The wound then heals, enclosing adventitious material between its surfaces, and corneal astigmatism is produced.

V.—ACUTENESS OF VISION.

After ordinary cataract operations three causes especially affect the visual acuity.

1. Secondary cataract.
2. Astigmatism of the cornea. We do not conceal the fact that this defect is the result of an inaccurate corneal incision in at least one half the cases.
3. Senile changes in the choroid.

VI.—SECONDARY OPERATIONS.

These are called for in (1) secondary cataract and (2) occluded pupil.

In the first instance I usually perform discision with a Graefe knife; occasionally, however, I use the discision needle. A narrow Graefe knife is passed into the anterior chamber through the lower segment of the cornea (about

2 *mm* from the limbus) until its point reaches the upper limit of the coloboma, and the cataract is divided down to the pupillary border. By this means, especially in recent cataracts, a wide opening is made in the membrane.

This procedure is productive of better visual results, and causes less bruising than discision with the needle. When several months or a year have elapsed since the extraction, it is always more difficult to make a gap in the secondary membrane on account of its increased density. For this reason I employ in such cases a simple needle or have recourse to the double-needle operation.

In my opinion discision can, in normal cases, be advantageously done as soon as three weeks after the extraction.

In explanation of the relatively small number of discisions for secondary cataract—fifteen out of forty-four cases—I would point out that most of the patients belong to the peasant class, who, as a rule, are satisfied with a small amount of sight, and hence do not take advantage of a secondary operation.

In one case only was an operation performed for the relief of occluded pupil—the first patient mentioned under heading II. A. An iridectomy with the Graefe knife was followed by excellent results.

Original vision.	Number of cases.	Improved to.						Total number improved.	Ultimate vision.		
		$\frac{6}{6}$	$\frac{6}{9}$	$\frac{6}{12}$	$\frac{6}{18}$	$\frac{6}{24}$	$\frac{6}{36}$		Good.	Indifferent.	Lost.
$\frac{6}{6}$	1								1		
$\frac{6}{9}$	7	1						1	7		
$\frac{6}{12}$	27	2	3					5	27		
$\frac{6}{18}$	61	1	8	6				15	61		
$\frac{6}{24}$	41		1	3	3			7	41		
$\frac{6}{36}$	35		1	2	5	2		10	35		
$\frac{6}{60}$	20				4	3	2	9	9	11	
$\frac{3}{60}$	1									1	
$\frac{2}{60}$	1				1			1	1		
$\frac{1}{\infty}$	4										4
0	1										1
Not tested.	1										
	200							48	182	12	5
										199	

ANOMALIES OF THE OCULAR MUSCLES.

[FOURTH PAPER.]

By Dr. GEORGE T. STEVENS, New York.

VII.

AN EXAMINATION OF VON GRAEFE'S DOCTRINE OF "ANTIPATHY TO SINGLE VISION."[1]

IN Von Graefe's classical description of the condition to which he applies the term, "Antipathy to Single Vision," he says: "It has sometimes happened to me that after squint operations with apparently correctly adjusted visual axes there have resulted double images only slightly removed from each other. The visual power of each eye has been quite good, alternating strabismus having previously existed, and the accommodative power has been similar in the two eyes. Nevertheless it has been in no way possible to bring about single vision."[2]

He states that this condition of double seeing persists notwithstanding the interposition of prisms in various positions, and that every effort of the patient to unite the images results only in removing them to still greater distances.

He adds: "The tendency appears to be a direct physiological contradiction; for while in sound eyes some approach, especially in case of large retinal pictures, is sufficient to induce arbitrary muscular contraction in the interest of single vision, in these cases, on the other hand, it is found that exactly the opposite condition prevails, there being an absolute incompatibility to single vision."[1]

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[1] Read at the meeting of the American Medical Association, May, 1891.

[2] *Arch. für Ophthalmologie*, i., 1, 117.

In a still later article[2] he describes the condition more at length, pointing out the fact that the double images, although not far removed, pass from one side to the other, above and below, or one behind the other, always on the point of uniting, but never united. Von Graefe explains the anomaly on the theory, first, of injury to the nervous centres or faulty projection from the retinæ, and, second, of the supposed difficulty of obtaining regular associated action between muscles which have long been unaccustomed to association.

A careful examination of many of these cases of "antipathy to single vision" leads me to conclusions different from those arrived at by von Graefe, and the practical application of these conclusions has shown that they are, at least in a great measure, correct. That, in a proportion of these cases, the technical details of disturbing elements may elude our search is undoubtedly true, but that the principles which I shall present will apply very generally I am fully convinced.

In a paper read before the American Medical Association, June, 1889,[3] I have already expressed my disbelief in the generally accepted view of the existence of a physiological antipathy to single vision, and my conviction that, by properly selected and properly executed proceedings, this unpleasant condition may usually be eliminated.

The view of the causation of this anomaly, as set forth in that paper, may be briefly condensed into a single sentence.

The condition of antipathy to single vision as described by Graefe and by subsequent authors depends not upon lesion of the brain or faulty projection of the images from the retinæ, but upon unequal tension of corresponding ocular muscles under the influence of corresponding nerve impulses directed to them.

I do not assert that no irregularity in projection from the retinæ can occur, nor that cerebral lesions may not induce incorrigible diplopia. Such cases, if found, will present other and more definite phenomena of the existing ocular or central nervous disease than simply the presence of diplopia. The proposition refers to the cases which come fairly within the class so clearly described and so sharply defined by Graefe.

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[1] *Arch. für Ophthalmologie*, i., 1, 118.

[2] *Arch. für Ophthalmologie*, iii., 1, 119.

[3] Published in ARCHIVES OF OPHTHALMOLOGY, vol. xviii., p. 371.

Excluding then, cases of paresis, and confining ourselves to the class of cases thus defined, we shall find the causation of this supposed antipathy mainly in two conditions. The first of these is acquired as the result of the squint operation or operations. It consists in the fact that by unequal settings back of the insertion of the corresponding tendons, there are induced irregular responses to the impulses directed to these corresponding muscles. In the examination of a very large number of cases, I have found this condition, but generally modified by a second to be mentioned presently, by far the most prevalent. There will be found in such cases either an excessive rotating power, or a power of rotation only moderately restricted, of the muscle of one eye, internal or external rectus, while the rotating power of its direct associate of the other eye is materially restricted. As a consequence of this irregular and unequally balanced condition of opposing muscles no harmonious adjustments can be made, and the more earnestly the patient strives to bring about associated action, the more signally he fails.

That "settings back" of the ocular muscles to an unequal extent may be made with propriety and with success, was taught by the great master, von Graefe, himself, and the practice has been very generally continued to the present time.

It is true that von Graefe advised in extreme cases a division of the operation between the two eyes, but in fact, this was a concession only to most obvious disabilities involved in the failure of the most conspicuously associated movements toward the side opposite the eye operated on, if in a case of converging strabismus, and an impediment to even approximate convergence at near points. Within the limits of extreme results a full correction of the appearance of squint was, according to his teaching, not only permissible but desirable. This proceeding of making a correction of squint by a single operation has been supposed to be sustained by the theory that the muscle of the squinting eye is pathologically shortened to a considerably greater extent than its antagonist.

It may be conceded that there is, within certain limits of unequal tensions of accordant muscles, a possibility of union of images; otherwise binocular vision would in no case be established except when exact balancing of the tension is secured. That easy and permanent associated action is compatible with any considerable degree of diversity of tension, I do not believe.

It may be objected that if this proposition is correct the proportion of perfect corrections for squint by the prevailing methods of operating must be less than is ordinarily supposed. It is probable that examinations made by methods more exact than those which have been ordinarily employed would show that a very large proportion of cases in which binocular vision has been presumed to have been established by the ordinary squint operation is much smaller than is commonly supposed. However this may be, whether the results of the standard squint operation is often successful or not, it is certain that such unequal tensions are in many instances incompatible with a blending of the images.

A second causative influence, and one which acts as an element in nearly all of these cases, is the difference in relative tension of muscles which act in the vertical direction.

The instinct of bringing images to the same horizontal plane appears to be very great and only second, if indeed it is second, to that of union of images. In case of double images, whatever the difficulty of blending may be, the patient instinctively exerts his best endeavors to place the images in the same horizontal plane. It thus happens, that in certain cases in which, either with or without slight lateral inequality of tension, the effort to bring images to the same plane induces a lateral squint,—a squint which may be variable; that is, such a squint may at one period of life be converging, and at another diverging. Thus, in a case now under my observation, the patient was, during her childhood, subject to a very pronounced converging squint. During the past twenty years she has had conspicuous diverging squint, although no operation has been made. Such a condition may properly be called *variable strabismus*, its character of divergence or convergence changing from time to time, remaining possibly in one direction during several years, then changing to the opposite direction.

If in such a case one or both interni or externi are cut, double vision of an intractable form is likely to result. In such cases we must make diligent search for the vertical deviation, a condition often obscure or not at all manifest, before we can succeed in establishing binocular vision.

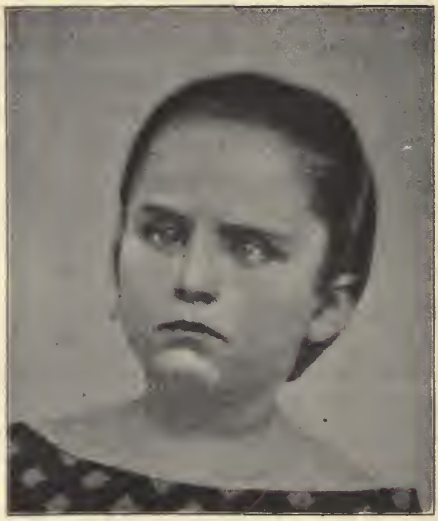

FIG. 1.

I have had the good fortune to obtain a daguerreotype portrait of the lady whose case has just been mentioned, taken during her childhood, and from which I have made an enlarged photograph which I have the pleasure of showing (Fig. 1). The photograph shows converging squint, so pronounced that few ophthalmic surgeons even at the present day would hesitate in regarding it as a proper case for a free division of the internus of one, if not of both eyes. Had such an operation been performed upon this patient in early life, intractable diplopia would doubtless have resulted, and eventually extreme diverging strabismus. In another photographic portrait of this lady, made by myself a year ago (Fig. 2), a moderate appearance of diverging strabismus is seen. In fact a divergence of more than 12 ° with corresponding crossed diplopia existed. But the most striking feature suggested by this photograph is the left hypertropia, well defined in the position of the eyes and of the brows. The question at once arises whether the change from converging to diverging strabismus is not the result of some radical change in the static condition of the eye-muscles. That this is not the case will be seen by the most cursory glance at the third portrait (Fig. 3), in which the old converging squint has returned. This condition of convergence can be induced at any time by the application of a solution of atropine to the eyes. At the time of taking this third portrait, atropine had been applied twice a day for three successive days, and its effects were complete. A week after

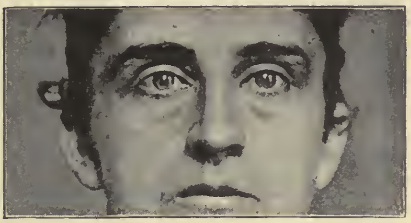

FIG. 2.

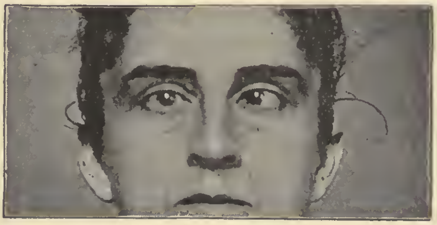

FIG. 3.

that time the diverging squint had returned and has continued during the year. The transformation from divergence to convergence occurs within an hour after the first application of the atropine, and the present habitual condition of divergence returns as soon as the effect of the drug passes off. This variableness is, without doubt, due to the action of the muscles in the effort to overcome the unequal height of the images, and until the condition of the hypertropia is eliminated such changes of the lateral relations are liable to occur. In passing, a single thought may be given to the adverse testimony of such cases to the well known doctrine of the relations between the excessive efforts at accommodation and converging strabismus; for here the converging strabismus occurs only when accommodation is neutralized. The present interest in the case centres in the fact that had a free tenotomy of an internus been practised during childhood, the almost certain result would have been an "antipathy to single vision." Even at the present time, when the conditions of hyperphoria and hypertropia are carefully sought for, this patient is able, under many circumstances, to conceal completely the evidence of hyperphoria as shown by the phorometer.

A curious fact in connection with the inequality of tension of the lateral muscles associated with hyperphoria is that a condition which in a passive state of the muscles of either eye or of both eyes is a marked divergence, becomes, as soon as all the muscles are engaged in the act of adjustment, pronounced convergence attended by homonymous diplopia. Conversely, a like change may occur when the tendency is, in the passive state, to converge.

A very practical illustration of what has been stated may be found in an actual history which I condense for this purpose. In a child with left hyperphoria (or hypertropia) the left eye drifted outward during the first few years of life. (Such a diverging squint is rarely observed by parents unless it is extreme.) At length, presumably as the result of a change in the methods of making the efforts to adjust for the horizontal plane, the squint became convergent. The appearance of the eyes of this patient in early childhood is represented in Fig. 4, which is copied and enlarged from a daguerreotype picture of him taken at the age of about three years.

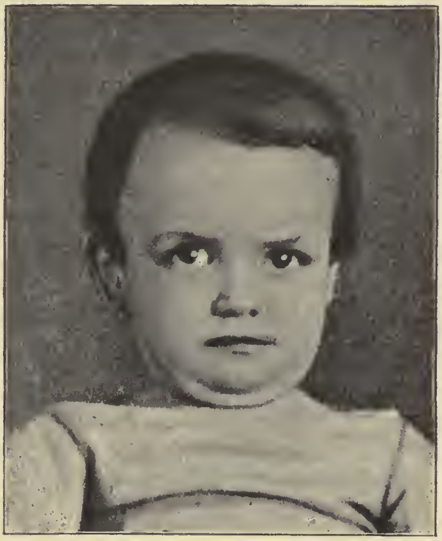

FIG. 4.

It is to be remembered that in the daguerreotype portrait the features are reversed.

About three or four years later, and after the divergence had given place to convergence, a surgeon performed tenotomies of the two recti interni, presumably severing both tendons completely. The result proving an over-correction, the effect of the operation on one eye was modified by the insertion of a (conjunctival?) suture. The result, in view of what has been stated above, was such as we might anticipate. Double vision of the most annoying character was always present. The efforts of many distinguished surgeons to induce these twin images to unite by means of prisms, even for an instant, were fruitless after trials repeated during many years. The case found a prominent place in ophthalmological literature, endorsed by several distinguished surgeons, as one of the class now under discussion. No case could therefore afford us a better model for our study.

It proved easier for this patient to fix objects with the left eye. That being the case, we may suppose that he would make a certain effort of adjustment with the weakened internus of that eye, which would be associated with a corresponding excessive effort of the opposite internus. But these unequal efforts would also be combined with the effort to adjust for the difference in the positions of the images with reference to the horizontal plane. The result would be an excessive rotation of the right eye to the nasal side with homonymous double images. If, on the other hand, a patient with this combination of defects should look at an object which is isolated from other objects which may serve to aid the disabled eye in active adjustment, as, for instance, at a church spire or at the moon or a bright star, in which cases only the clear sky is in immediate relation to the object, the most disabled eye would at once drift outward, and crossed diplopia would result. So also, if a small screen were to be interposed between either eye and the object looked at, thus shutting it out from active participation in the effort of adjustment, the covered eye would diverge in a marked manner and would move inward, perhaps to the extent of one third the diameter of the cornea, as soon as the screen should be removed, and then homonymous diplopia would be observed. Or again, if the subject of such a defect were to look through a grating, as, for instance, through the palings of a picket fence, an object seen beyond the fence would be seen doubled, and the diplopia would be crossed; while an object seen between the person and the fence would be double also, but the double images would be homonymous. In the first instance, looking beyond the fence, the adjusting energy of one eye becomes passive, as in the case of looking at the object against the clear sky, with resulting crossed diplopia. In the other instance, looking at the object nearer than the fence, active adjustment of both eyes occurs with excessive convergence and homonymous double images. All these suppositions and conclusions have been fully justified in the actual treatment of these cases.

In certain cases in which this intractable diplopia exists, the patient has the extraordinary faculty of selecting at will which eye shall fix the object, and the diplopia becomes homonymous or crossed, according as the one or the other eye is engaged in direct fixation. Thus, in the case of a lady patient, if I direct her to look with the right eye, I can see that the eyes are strongly converged, and she reports the presence of homonymous double images. If I then direct her to look with the left eye, the change may require a few seconds or it may take place very quickly. Then the eyes are plainly seen to diverge, and the diplopia is crossed. In such a case, the nervous impulse directed to the right internus is greater than the normal during fixation with that eye, and when a corresponding impulse is sent to the left internus, the eye is caused to swing in excessively and too great convergence results. On the contrary, when the left eye is fixed, there is no such excess of nervous impulse sent to the left internus, and as no excessive impulse is therefore sent to the right internus, that eye swings outward passively, and crossing of images results. But in this explanation we must not lose sight of the important, probably the most important, element in the causation of the inward swing in many if not in the majority of cases. This is the influence of the difference of tension of the muscles which move the eyes in the vertical direction. This influence is often sufficient, even though the moving power of the corresponding lateral muscles should be equal, to cause a swing beyond that which would result from a given impulse to a lateral muscle. I have rarely met with a case of the so-called "antipathy to single vision" in which this element did not play an important rôle.

It needs no argument to show that the defect which causes the patient to have homonymous diplopia when looking at an object, say at twenty feet distance, and heteronymous diplopia when looking at the same object, if a grating, like the fence of which we have spoken, is interposed between the eyes and the object, or which causes the same patient to have homonymous images when looking at an object surrounded by other objects, and crossed images when looking at an object isolated from visible surroundings, lies neither in the brain nor in faulty projection from the retinæ, but in peculiar, although perhaps obscure, defects in the adjusting apparatus.

Homonymous diplopia caused by a brain defect or a retinal defect would remain homonymous under all the circumstances which have just been mentioned. Activity or passivity of certain efforts of adjustment could and would be modified by the circumstances mentioned, and the phenomena resulting, which at first appear confused and irregular, are in reality uniform and in accordance with fixed laws.

It is not to be denied that the investigation of an individual case is often surrounded by immense difficulties, or that each new case may present new and perhaps at first incomprehensible phenomena. It is none the less true that all these cases will be found to be governed by known laws. If the difficulties of interpretation of the phenomena in individual cases are great, it demands the greater skill and patience on the part of the investigator in searching out all the various influences which combine to make the defect.

It would probably be impossible, even if all the facts were in our possession, to lay down distinct rules for the discovery of the exact nature of the faulty relations of muscular tension in each case of this class. To attempt it even to the extent to which the principle has been studied, would be to record separately the experience in each individual case. Enough has been said, however, to indicate the lines on which the investigation is to be made.

In the correction of the defect,—this incompatibility to single seeing,—we must logically seek to re-establish a degree of equilibrium between the mobilizing functions of the opposing muscles. To this end, after first correcting the unequal tendency in the vertical direction, it will, in most instances, be necessary to bring the insertion of the disabled muscle forward, and to reduce the tension of the corresponding one of the other eye. This procedure is by no means as easy as might appear. To readjust the tendon after such a setting back as occurs in the standard squint operation, requires in many instances the most persistent and skilful treatment.

Very little can be accomplished by a gross advancement in which the already disabled muscle is made even less flexible than before. The object is secured by progressive steps, one slight advancement preparing the way for another, until the proper degree of rotation with as great flexibility of the muscle as possible is secured. Then the corresponding muscle of the other eye, if it has not been already relaxed, must be made to assume, as nearly as is required, a corresponding adjustment by a careful relaxation.

Such a procedure has, in my hands, succeeded in establishing binocular vision in many inveterate cases of the so-called "antipathy to single vision." The process is often long and difficult, making the greatest possible demands upon the skill, courage, and patience of the surgeon and of the persistent and intelligent co-operation of the patient.

It has been my purpose to do no more than to indicate in a very general way the principle which I would advance, namely, that incompatibility to single seeing usually denotes incorrect methods of operating, and that there can be no effectual correction of strabismus which leaves the tension of the corresponding muscles of the two eyes unequal. The supposed success of many cases in which such inequality is caused by operation, doubtless results from the fact that in the remaining diplopia the images are so widely separated that the patient does not become aware of its existence.

In order to avoid the evil results of diplopia and to secure not only possible but easy binocular vision, then, we must be guided, first, by the actual rotations of the eyes, and by the actual relation of the visual lines not only in the lateral but in the vertical direction. This can be accomplished only when the patient recognizes the position of double pictures, and this according to the theory of identity. There should be no final operation for the adjustment of squinting eyes, until both patient and surgeon are sure of the relative position of the double images.

A precept which I have elsewhere brought forward is in this connection also of essential importance, viz.:

"*Every modification of the length of a lateral muscle, by relaxation or by shortening, which can in any degree effect the rotation of one eye, should in every instance be accompanied by an exactly equivalent modification of the corresponding muscle of the other eye.*"[1]

If this precept is correct, the practice of relaxing or setting back of an internus, accompanied by an advancement or shortening of the externus of the same eye while the muscles of the other eye undergo no modification, cannot afford the best results.

The subject of the determination of the relative tensions of the various eye-muscles I have already discussed at some length in a series of articles published in the ARCHIVES OF OPHTHALMOLOGY during the years 1887, 1888, and 1889. It is unnecessary to repeat what has there been said, but there is also much more which might be said in connection with the examination of the relative tensions in these peculiar and most difficult cases, which must be reserved for another occasion.

One point, however, is so important in its relation to the subject under consideration that it should not be separated from it. This is the examination of the comparative rotation of the two eyes in the temporal and in the nasal direction. Something can be learned from an inspection of the movements of the eyes as the surgeon causes the patient to follow with the eyes a pencil or other object as it is carried from side to side, the patient making his best efforts to see it as far toward the extreme outer field of vision at each side as possible. It will in most of the cases, in which the original operation has been performed for converging strabismus, be seen that as the eye on which the operation, or the most extensive operation, has been performed moves toward the nasal side its progress is arrested before the border of the cornea reaches the inner canthus.

The nasal rotation of the other eye will often be found to be free or even excessive. But even the nasal rotation of this other eye may be restricted, only in less degree than the first. Similarly we may observe a difference in the temporal rotation. This method of inspection, while it is important and in some measure satisfactory, furnishes less exact information than is required in most cases.

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[1] Anomalies of the Ocular Muscles, ARCH. OF OPHTHALMOLOGY, 1889, p. 407.

In the measurement of the temporal rotation we have in the perimeter an instrument of sufficient precision. It is my custom to place in the carrier of the perimeter a few capital letters in clear type, of about the size, 2 = D, of the Snellen scale, and require the patient to see the letters with sufficient clearness to read them. A glass to meet the requirements of the patient's vision is held in such a way before the eye that it is at a right angle to the line between the eye and the test object. Ordinarily a pair of eyes in which no considerable degree of heterophoria exists will make the temporal rotation about 40° each, and no considerable difference in this rotation should exist. In many of the cases under discussion the temporal rotation will be found to differ for the two eyes to the extent of from 10 to 20°. With the new model of the registering perimeter this element can be quickly and accurately determined.

Unfortunately we have no method so accurate for the nasal rotation. The full rotation of the eyes in this direction in the normal condition cannot usually be determined by the perimeter, except by the supplementary use of a strong prism. Even with this supplement, the extreme rotation inward of cases of slight converging squint cannot be registered by the instrument. In cases in which the rotation does not much exceed the line of vision across the bridge of the nose, we may place a rounded prism with its apex in the canthus. Two degrees of prism equals one degree of the perimeter. Hence a prism of 10°, apex in the canthus, may cause the eye to turn inward 5° more than it would if looking directly at the object. The expedient of placing a scale of degrees on the lower lid and reading from it the nasal rotation, while inaccurate, is sometimes the only recourse, and the best use of an unsatisfactory method must be made.

THE METASTATIC TUMORS OF THE EYE.

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(With six figures on Plate III. of vol. xxii. of German Edition.)

THE primary tumors of the eye, because of their relative frequency, have been carefully described both clinically and anatomically, and are extensively treated of in the literature. The case is otherwise however with secondary tumors. The older ophthalmologists and many of the recent text-books speak only of the direct encroachment of such malignant tumors as have arisen in the vicinity of the eye, and it is only of late that the metastatic tumors of the eye have been noticed. Since these tumors are of interest not only from a pathological standpoint, but also from that of the clinician and general practitioner, I shall mention the cases of metastatic tumors of the eye which have been previously observed, and add an account of two cases which I have myself examined anatomically.

I.—METASTATIC TUMORS OF THE OPTIC NERVE.

I find very brief notices of these cases in the literature. Dittrich[1] described a case of multiple cancerous neoplasms, in which besides numerous tumors in the brain and various cerebral nerves, the optic nerves were found to be swollen just before their passage through the foramen, and the nerve-bundles were infiltrated with cancerous masses. The case of Jacobson [1] possibly belongs here, since there was a tumor of the intraocular end of the optic nerve consecutive to a tumor in the orbital tissue and corresponding to it in structure, being a myxosarcoma with sclerosis, ossification and hyaline degeneration (probably cylindroma [2]). Whether this tumor were in reality secondary, cannot be said with certainty.

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[1] *Prager Vierteljahrschr.*, vol. xii.

Krahn [3] reported a case where there was metastatic cancerous formation in both optic nerves just behind the lamina cribrosa, with carcinoma of the ovaries. The neoplasms lay between the nerve sheaths, sending the cellular masses of which they consisted between the nerve-bundles, and compressing the central vessels. The patient became almost entirely blind about eight months before his death, and three weeks before he died, the usual picture of choked disc and neuritis with hemorrhages in the discolored tissue was observed.

Schiess-Gemuseus and M. Roth described a metastatic sarcoma of the papilla and the neighboring retina.[4] Microscopic examination showed a spindle-celled sarcoma springing from the papilla and encroaching upon the retina, similar in structure to other metastases and to the primary growth—a nævus of the sternum which had undergone sarcomatous degeneration.

(A case of metastatic carcinoma of the papilla, though not a pure one, will be reported among the cases of metastatic carcinoma of the choroid, right eye. Case 3.)

The rarity of these observations makes the following case one of interest, although the clinical course of the disease was not followed with the care which might be wished.

Karl K., æt. forty-three, was received into the general hospital September 19, 1888, where the diagnosis "tumor renis dextri" was made, and where he was treated until his death. A few days after his admission he suddenly became blind in the left eye.

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[1] *Graefe's Archiv.*, vol. x., 2.

[2] *Cf.* Leber, in "Graefe and Saemisch," vol. v., p. 911.

[3] Reference in *Zehender's Klin. Monatsbl.*, vol. x., p. 103.

[4] *Graefe's Archiv.*, vol. xxv., 2, p. 177.

The ophthalmoscope showed a pale disc, and distended and tortuous retinal veins.

The patient died October 20, 1888, no change having occurred in the vision of the eye that had become blind. Autopsy: Inner meninges somewhat thickened, the left optic nerve showing an ampulla-form swelling at its entrance into the foramen, and being replaced by a gray vascular substance, sharply limited at the end toward the chiasm; carcinoma renis dextri primar., cum metast. pulm., pleur., gland. retroperiton. et renis sinistri (carcinoma metast. nervi optici sinistri ?)

The optic nerves were removed together with the eyes. On the intracranial segment of the left optic nerve just within the optic foramen there was a gray translucent club-shaped swelling, limited by a constriction toward the foramen, while toward the chiasm there was no such sharp limitation. The length of the tumor was about 10 *mm*, and the diameter of the nerve in the thickest part of the tumor 8 *mm*. In the optic canal the nerve was very thin, but 12 *mm* from the peripheral margin of the tumor it regained its usual thickness, and was here quite normal (Fig. 1).

Eyeball and nerve were hardened in Müller's fluid and alcohol, and embedded in several pieces, then cut transversely and longitudinally.

The sections of the nerve appeared normal macroscopically as far as a point 17 *mm* behind the lamina cribrosa; from this point posteriorly an increasing number of yellow dots were seen in the otherwise normal nerve-bundles. 30 *mm* from the ball, and 3.5 *mm.* behind the thinnest portion of the nerve, the tumor began as a round mass in the medial inferior part of the club-shaped swelling of the nerve, its yellowish-white smooth tissue being sharply defined from the meniscus-shaped nerve which surrounded it.

Microscopic examination.—The tumor consists of a well developed connective-tissue framework with numerous spindle-shaped and oval nuclei, and blood-vessels, and the meshes are filled with closely-crowded but relatively few large epithelial cells, having a finely granular protoplasm and a large nucleus with 1-2 nucleoli (Fig 4).

While the meshes in the centre are narrower and the epithelial nests compressed and irregularly grouped, the arrangements in the peripheral portions, especially outward and upward, is much more regular and the connective-tissue septa run parallel to the long axis of the nerve, giving the impression that the interfascicular septa have proliferated and tumor-tissue taken the place of nerve-fibres. Besides the epithelial and connective-tissue elements there are two other components of the tumor, one cellular and one amorphous.

Scattered among the connective-tissue bands of the tumor is a granular tissue in which lie irregularly-angular darkly-stained cells which seem to indicate a proliferation of the glia-tissue of the nerve.

In the peripheral portions of the tumor appear clumps of an amorphous substance staining a bright red with eosin, and brownish-black with Weigert's stain, arranged regularly along the coarser connective-tissue septa, compressed between the two heterogeneous tissue-elements of the tumor. The arrangement of these masses and their color-reactions would indicate that they are a corneous derivative of the medullary substance, which is being broken down by the pressure of the neoplasm.

Inferiorly the tumor reaches to the inner sheath of the nerve and on either side between the tumor and the nerve are only atrophic bundles of nerve-fibres which above become nearly normal. Only at the peripheral end can the nerve-bundles be followed a short distance into the tumor. In a similar manner at the central end the neoplastic tissue pushes into the nerve (Fig. 3), while in the entire longitudinal extent the limit between the two tissues is sharply marked. The nerve-bundles are compressed, the fibres irregularly arranged, and corpora amylacea are scattered through that portion of the nerve which is in contact with the tumor.

The optic nerve more centrally is œdematous, increased in diameter, the nerve-bundles loosened and enclosing spaces filled with finely-granular eosinophilous masses, while the peripheral bundles are atrophic. Nearer the ball the nerve shows some small localized atrophic foci but is otherwise normal. There has been no proliferation of the interfascicular tissue, but the pial sheath is vascular and the meshwork of the arachnoid sheath has been transformed into a dense cellular connective-tissue.

All the morbid changes disappear nearer the eyeball and the nerve for a distance of 17 *mm* from the ball is normal, excepting for some slight perineuritic changes.

The central vessels are distended with blood, but the papilla is unchanged.

This was then a case of metastatic carcinoma of the optic nerve developing in the intracranial segment near the optic foramen, destroying the nerve to a slight extent, but for the most part merely compressing it. The inner sheath of the nerve has not been perforated by the growth. The tissues of the nerve are partly atrophic from pressure, and this atrophy has continued downward in the form of irregularly distributed linear degenerations of single portions of the nerve-fibre bundles, while centrally the nerve is œdematous but not atrophic. The pressure of the tumor has caused a chronic inflammation of the internal sheaths of the nerve, which has extended downward to the entrance of the central vessels. It is interesting to see that notwithstanding the complete obliteration of the subvaginal space at the central orifice of the optic foramen, no hydropic changes are found in the nerve and no choked disc has developed.

Clinically only a paleness and discoloration of the disc with dilatation of the larger vessels were noted. As to diagnosis, a metastatic tumor of the nerve (*vid.* case 3, right eye, in following section) like a primary one would cause a disturbance of vision early, and rapid blindness with no ophthamoscopic changes in the beginning, and in case the metastasis were in the orbital portion of the nerve there would be disturbance in the mobility of the eye, and exophthalmus.

Besides the cases mentioned of metastases in the optic nerve trunk and papilla, the only others which have been studied clinically and anatomically are metastatic carcinomata and sarcomata of the choroid, which will be treated of separately.

II.—METASTATIC CARCINOMA OF THE CHOROID.

To the seven[1] cases which have been reported I would add another which was examined both clinically and anatomically.

T. Magdalena, æt. 57, was admitted to the eye clinic December 10, 1887, with a history of failing vision in the left eye for five weeks. February 18, 1887 the left breast was amputated for carcinoma and the enlarged axillary glands removed. Later a small swelling appeared in the wound and the axillary and supraclavicular glands became swollen, and the patient suffered with a severe cough and pain in the back and legs.

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[1] Abstracts of these cases may be found in the paper by Mitvalsky. These ARCHIVES, vol. xx., No. 2, 1891.

The left eye, normal exteriorly, showed a slight undulating detachment of the retina above the disc, of a pale grayish-yellow color. There was no perforation. The macular region was not detached, and the fovea was seen as a dark-red spot. Below the disc the retina was detached but of normal color. Tension normal. V=fingers at seven feet. Field of vision narrowed upward ; downward a large paracental scotoma. Right eye normal.

In the course of a few days the detachment of the retina increased until it surrounded the disc, and the vision grew worse while the tension remained the same. January 4th the retina was totally detached. Paralysis of the left recurrent laryngeal nerve developed, there were girdle pains in the region of the third and fourth ribs, increasing dyspnœa with catarrhal symptoms in both lower lobes, and the patient lost strength.

Later there were symptoms of spinal compression. The intraocular tension became increased, there was episcleral congestion, and the anterior chamber became shallow. The glaucomatous condition increased up to the death of the patient, March 28th, from exhaustion. The course of the retinal detachment—beginning in the upper half and extending—the increased tension, and, finally the appearance of glaucomatous symptoms in the anterior segment, caused a probable diagnosis to be made of *detachment of the retina by a carcinomatous metastasis in the choroid.*

The autopsy showed carcinomatous tumors in the brain, lungs, and liver.

The eyeball was hardened in Müller's fluid and alcohol and divided horizontally. The lens and iris were displaced forward, the retina completely detached, leaving a small conical space filled with shrunken vitreous. The entire subretinal space was filled with a homogeneous fibrinous mass. The choroid for 10-13 *mm* about the disc had a thickness twice that of the normal sclera. The thickened portion was of a mottled light-brown color, almost smooth, and sharply defined from the dark atrophic unthickened choroid.

Microscopic Examination.—The thickened portion of the choroid is transformed into a dense fibrous connective-tissue with spindle-formed and pigmented branched cells and a few blood-vessels, enclosing nests, tracts, and tubules of small epithelial cells which in their arrangement may be divided into two classes. The first are regular groups of cells, tubular in longitudinal section, in transverse section circular, surrounded often with a fibrous sheath, and always separated from the surrounding tissues by a basal membrane. Many of these contain cavities filled with degenerated matter or red blood corpuscles. The arrangement is most regular in the layer corresponding to the chorio-capillaris. In Fig. 6 lumina are seen which on one portion of the wall have a single layer of cells like endothelium, while in other portions there are several layers of cells showing transition forms.

Beside these there are lumina which have a regular laminated lining, and finally solid cell cylinders. Nowhere can a trace of the vessel endothelium be seen between the vessel wall and the lining epithelial cells. Connected more or less directly with these are seen epithelial cells arranged in a second manner—tracts and groups of cells, which, not surrounded by a basal membrane, seem to force their way into the connective-tissue stroma, are always solid, and here and there show signs of retrograde metamorphosis.

A few vessels are found in all layers of the tumor, but more numerously in the inner, part of them narrow with thick walls and part wide with thin walls like the normal capillaries of the inner choroidal layer. Many of these vessels are partially occluded by hyaline deposits on the lining endothelium.

The lamina vitrea has been destroyed in many places, and the pigment epithelium is irregular and partly wanting.

The two superior venæ vorticosæ, which were examined in transverse sections, were occluded partly by proliferated endothelium, partly by epithelial groups lying in masses of coagulated blood, portions of which had undergone calcareous degeneration.

All portions of the tumor, but chiefly those which are apparently the oldest, near the papilla, show evidences of retrograde metamorphosis, and these portions do not take the stain. Some parts also have undergone calcareous degeneration. The tumor is sharply limited equatorially from the uninvolved portion of the choroid, which is markedly atrophic, and contains a few vessels with sclerosed walls. The iris, which is pressed against the cornea in most of its extent, shows similar changes. The choroid surrounds the papilla as usual. The nerve and retina show but slight changes. The iris is adherent to the cornea at its periphery. There is an increase in nuclei about the dilated episcleral vessels.

Comparing the condition in this case with that found in some other cases reported, the marked features common to them all are the alveolar structure of the flat diffuse carcinoma and the presence of carcinoma cells in the vessels. In one case the carcinoma cells seemed to replace the endothelium of the vessels, furnishing a new proof of the metastatic development of endothelium into epithelium,[1] caused by the epithelial infection of the former with the particles of carcinoma carried in the blood. At any rate, we may safely say with Perls that the metastases in those cases are caused by carcinomatous emboli in the choroidal vessels; the alveolar structure is due to the fact that the epithelial masses develop first in the blood-vessels at the point of invasion and spread for a time within the vessel, then break through the walls and spread atypically, causing hyperplastic tissue-changes in the stroma of the choroid. The occlusion of so many vessels leads to atrophy of the rest of the uveal tract and to retrograde changes in the growth itself.

Taking the eight cases together, some interesting facts reveal themselves. The primary growth was located six times in the mammary gland, once in the lung (in a male), and once in the stomach.[2]

All the cases in which an autopsy was made showed numerous metastases in various organs, and always in the liver and lungs or pleura.

Passing Case 3 in which the starting-point of the neoplasm was the nerve, carcinoma of the choroid has appeared in two distinct forms. Twice there has been seen a circumscribed elevated tumor which has quickly destroyed vision. In all the other cases there has been a flat, slow-growing tumor, not causing much visual disturbance until the retina has become detached. This form can be diagnosed with the ophthalmoscope with certainty only before an extensive detachment has occurred. In the cases examined sufficiently early, either yellow or reddish-white spots were seen, or flat tumors showing irregular absorption of the pigment epithelium.

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[1] *Vid.* Klebs, "Die allgem. Pathol.," vol. ii., pp. 504 and 771, 1889.

[2] The author refers here to Gayet's case of adenoma of the choroid, not included by some in the category of pure carcinomata of the choroid.—TRANSLATOR.

To repeat, IN *these cases it is certain that the metastases take place through the medium of the blood-current and that the carcinomatous growth develops first or possibly entirely within the vessels of the choroid.* The location of the growth corresponding to the point of entrance of the short ciliary arteries into the choroid, and its diffuse development in the choroid without involving neighboring parts, strongly support this view.

III.—METASTATIC SARCOMA OF THE CHOROID.

Metastatic sarcoma has been observed far more rarely.

The first case was reported in 1870 by Brömser.[1] One year after the removal of a pigmented tumor which bled readily, having developed from a congenital pigmented nævus, a melanotic sarcoma appeared in the choroid, without a local recurrence of the tumor.

Leber (*l. c.*), who examined the enucleated eye microscopically, considered the tumor metastatic, notwithstanding the objection of Fuchs.[2]

Pflüger[3] described a unilateral metastatic sarcoma of the choroid following a pigment nævus of the right clavicle in a woman æt. thirty, after it had become sarcomatous. The second eye was amblyopic, but no ophthalmoscopic examination was made and no anatomical examination of the tumor.

Notwithstanding the fact that the cases reported of metastatic sarcoma are so few, the relative immunity of the choroid from metastases which Fuchs[4] held, and the statement of Virchow,[5] so often quoted, seem to be disproved. It is questionable further whether, in some of the cases in which a sarcoma of the choroid has been considered primary, it were not in reality metastatic. This applies particularly to those cases in which, after the early removal of a strictly localized melanotic tumor of the choroid, the patient dies from metastases in internal organs; and to those in which tumors appear at different times in the two eyes.

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[1] Ueber einen Fall von secundärem Melanom der Chorioidea, *Inaug.-Dissert.*, Berlin, cited by Leber, *Arch. f. Ophth.*, vol. xxxi., 4, p. 111.

[2] "Das Sarcom des Uvealtractus, 1882, p. 238.

[3] *Arch. f. Augenheilk.*, vol. xiv., p. 129.

[4] *L. c.*, pp. 201, 231.

[5] "Die krankhaften Geschwulste," vol. i., p. 69.

So long as the uveal tract was considered the only location of melanotic sarcoma,[1] and the possibility of the formation of metastatic sarcoma in the uveal tract was excluded, all the cases observed, even those varying from the usual course and aspect, were necessarily placed in this one category. But since the incorrectness of these views is proven, and since many intra- and extra-bulbar melanomata have been observed, which throw suspicion on the common idea of the frightful malignity of sarcoma of the uveal tract, perhaps a number of these cases can be more logically explained by the supposition that *the tumor in the eye is but a metastatic manifestation of a general sarcomatous infection*, whose starting point is to be sought for outside the eye. So long, however, as careful investigations are wanting, and so long as the literature of clearly secondary sarcoma of the uveal tract is so scanty, we can only advance hypotheses, which may serve the purpose of attracting more attention to this point in the future.

Explanation of the Figures.

Fig. 1. The tumor of the optic nerve seen from above (natural size).

Fig. 2. Transverse section through the middle of the tumor (Weigert's stain, slight magnification).

Fig. 3. Longitudinal section through the central end of the tumor (magnification as in Fig. 2).

Fig. 4. The structure of the carcinoma in the middle of the tumor (hematoxylin-eosin stain; Reichert obj. 9, eye-piece III.; drawn with drawing-prism).

Fig. 5. Horizontal section of left eye after the removal of the fibrinous mass filling the subretinal space (natural size).

Fig. 6. Transverse section through the tumor, from the upper half of the ball (hematoxylin-eosin stain; Reichert obj. 5, eye-piece I.; drawing-prism).

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[1] von Wecker in *Graefe-Saemisch*, vol. iv., p. 631.

A CONTRIBUTION TO THE STUDY OF THE TUMORS OF THE LIDS.

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Translated by Dr. Ward A. Holden, New York.

(With Fig. 1–10 on Plates IV. and V. of vol. xxii. Germ. Ed.)

ADENOMA.

OF all the neoplasms of the lids, the adenomata have the greatest interest for the histologist. There are few organs which within so narrow an extent are so rich in different glandular structures as the lids. It is therefore not surprising that the adenomatous growths show great variations in structure. Few cases of this sort, however, have been observed and described. I shall report here three cases of adenoma, each of which has developed from a different variety of gland. The preparations are from the collection of Prof. Fuchs, to whom my thanks are due.

I.—ADENOMA OF THE MEIBOMIAN GLANDS.

Antonie P., æt. 80, laborer, was received into the clinic January 5, 1887. She states that a year before she noticed an elevation the size of a pin-head in the middle of the left upper lid.

The left upper lid is now almost entirely involved in a pendant tumor the size of a walnut, which overhangs the palpebral fissure (Fig. 1). The tumor at some points is as hard as cartilage, at others softer; the surface is in some portions smooth, in others roughened; the color is somewhat yellowish. The skin is not movable over the tumor. The ciliary margin of the lid as far as the outer third, is involved in the tumor, thickened, and without cilia. The lymphatic glands in the neighborhood are not enlarged.

The tumor was removed January 7th, and the wound covered with a quadrilateral skin-flap from the cheek. Healing normal.

The extirpated tumor was hardened in Müller's fluid. It involved the inner two thirds of the lid, being 3.5 *cm* in its greatest height, and 2 *cm* in its greatest thickness.

Sections were made through the middle of the tumor, and also through its outer portion, where the margin of the lid was still in part preserved. The latter sections will be described first, since they are more important in explaining the relations of the tumor than the others.

The skin shows no particular changes. The subcutaneous tissue is thickened and transformed into a fairly tense layer with parallel fibres, containing normal sweat-glands here and there, and a few obliquely-cut hair-follicles. It is not sharply distinguished from the capsule of the tumor, which immediately underlies it. Widely distended veins mark the limit between the two.

The conjunctiva is thickened, vascular, infiltrated with round cells, and shows hypertrophied papillæ over the tumor, but it is only slightly infiltrated over the normal portions of the tarsus.

The tarsus shows the most marked anatomical changes. It is considerably thickened in its upper portion. The Meibomian glands are normal in the lower portion of the tarsus, further up these are compressed and small, and still higher the acini are replaced by lobules of the tumor, which at the sides lie in the normal tarsal tissue, and in the middle pass over into the mass of the tumor. The tumor fills the space between the skin and tarsus above the arcus tarseus, in which region it completely replaces the muscle-fibres and intermuscular tissue.

The tumor proper consists of an aggregation of irregularly formed, branched, anastomosing lobules, separated by connective-tissue septa, the whole enclosed in a connective-tissue capsule (Fig. 2).

The lobules vary in size, exceeding the normal acini of the Meibomian glands ten to twenty times; they are composed of a mass of round or oval cells, mostly polygonal from pressure, with no intervening substance or supporting tissue and no blood-vessels. The cells resemble the peripheric cells of the normal acini in the relation of nucleus to body, and in their staining properties, although their form and arrangement are irregular.

The central portions of the large lobules show retrograde metamorphosis. The nuclei do not stain, the protoplasm is granular, the margins of the cells become indistinct, and finally the whole degenerates into a granular irregularly-cleft mass, in which here and there a bit of nuclear structure can be distinguished.

Besides this fatty degeneration, other portions of the tumor show a degeneration independent of the former, involving whole groups of lobules with their septa. The nuclei of the epitheloid cells do not stain here; and the connective-tissue cells stain faintly, while their contours remain sharp.

The septa (Fig. 3, *s*) consist of tense parallel connective-tissue fibres with scattered oval or grouped round cells. The periphery of the tumor is covered with a capsule, whose structure is similar to that of the septa. Vessels are rare.

Between the lobules and the septa there are scattered cavities, for the most part empty or containing a few angular cells or amorphous masses. These cells have a different aspect from those of the lobules, their nuclei are smaller, stain less deep and are relatively smaller as regards the cell body. Where the section has passed through both tissues—the epithelial mass composing the lobule, and the septum,—the boundaries of the two tissues toward the cavity are very sharp, and endothelioid cells, some flat, some prominent, lie on either surface (Fig. 3, *e*). These cavities must then be considered lymph spaces, the analogues of those surrounding the acini of the normal Meibomian glands.[1]

In its central portion the tumor has involved the thickness of the lid from skin to conjunctiva, and the degenerative processes are here more advanced.

From the microscopic examination we may make the following deductions as to the origin and development of the tumor:

It appeared first in the upper portion of the lid internal to the middle line. From this point a part of the tumor spread downward and outward, while the principal mass perforated the tarsus and spread subcutaneously upward over the fornix, and also downward and forward in the intermuscular tissue, which, as well as the fibres of the orbicularis, it has almost entirely replaced. The tumor extends outward over the normal portion of the tarsus, but lies further distant from the margin of the lid, leaving a small bit of margin near the outer canthus free.

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[1] Czerny, *Klin. Monatsbl. f. Augenheilk.* vol. xii., p. 422, 1874, and Fuchs, "Zur Anatomie der Blut und Lymphgefässe der Augenlider, *Graefe's Arch. f. Ophth.*, vol. xxiv., 3, p. 1.

Taking into consideration the location of the tumor and its glandular character, there can be no doubt as to the diagnosis. The acini filled with cells and having a lumen only where the central portions have degenerated, are of the type of Meibomian glands. This view receives its chief support, however, from the evident transformation of the normal acini into lobules of the tumor.

We can speak with much less certainty as to the clinical nature of the tumor, since the benign character shown by the microscopic examination contrasts strongly with the rapid development of the tumor. In this regard the case presents a marked variance from those previously observed, in which the tumor developed much more slowly and did not attain so great a size. There have been reported, however, but two cases (Baldauf[1] and Bock[2]), which were proven to be adenomata of the Meibomian glands. In Fuchs'[3] case it could not be determined with certainty whether the tumor had originated in the Meibomian or the sebaceous glands.

In these two latter tumors as in our case there was the characteristic peculiarity that the lobules were for the most part solid, and only contained a lumen after the degenerated products of the cells had collected in the interior. The sharp limitation of the septa from the epithelioid contents, which was almost without exception the rule in our case, seems not to have been found in Bock's case, and cannot therefore be regarded as characteristic for this class of tumors. I limit myself to these remarks, since it would be useless to attempt to sketch a general picture of a variety of tumor from a consideration of but three cases, and I shall only add that in all these cases the patients were women of advanced age.

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[1] Baldauf, "Ein Fall von Adenom der Meibomschen Drüsen," *Inaug.-Dissert.* Munich, 1870.

[2] Bock, "Ein Fall von Adenom der Meibomschen Drüsen," *Wiener klin. Wochenschr.*, 1888, vol. i., p. 799.

[3] Fuchs, "Ueber das Chalazion und einige seltenere Lidgeschwülste," *Arch. f. Ophth.*, vol. xxiv., 2, p. 121.

II.—ADENOMA OF THE TARSAL GLANDS OF KRAUSE (ACINO-TUBULAR GLANDS).

Sylvester D., æt. sixty-five, came to the clinic in March, 1887, with a tumor of the left upper lid which springs from the conjunctival surface of the upper portion of the tarsus about the middle of the lid, and arches the tarsus outward in a saddle form. The margin of the lid is normal. The tumor presents itself in the palpebral fissure as a mass the size of a cherry, of dark red color, its surface rough and uneven, and bleeding readily. The base of the tumor is slightly constricted and covered with conjunctiva. The ball and the remaining adnexa are normal (Fig. 4).

The tumor was removed together with a bit of the neighboring tarsus, and hardened in Müller's fluid.

The portion of the tarsus next the tumor is compressed vertically and distorted so that in a vertical section the ducts of the Meibomian glands are cut obliquely or even tranversely. The capsule of the tumor is a direct continuation of the tarsal tissue which splits and encloses the tumor, being interrupted on the convex anterior surface, where the conjunctiva is also wanting and the degenerating tissue of the tumor is exposed.

The tumor consists of a number of branched and anastomosing glandular tubules, separated by connective-tissue septa. These tubules are of varying form and size, and consist of a smooth or folded epithelial wall enclosing a lumen of varying width. No membrana propria could be discovered.

The greater number of the tubules are thick and have many branches. At some points, however, there are groups of simple tubules representing the most elementary type of adenoma.

The epithelium of these tubules forms a wall of a single layer of cubical or cylindrical cells with oval nuclei, enclosing a considerable lumen. In most cases even the smallest tubules show here and there a second layer of cells which, lying on the inner surface of the first layer, cause an irregular thickening.

These cells with a rounder nucleus represent the first formation of the folds in the epithelial layer, which are so excessively developed in the older tubules (Fig. 5, c). These show numerous teat-shaped epithelial processes, consisting of two rows of cells, not supported by connective-tissue, springing out into the lumen of the tubules, and in places almost occluding them (Fig. 5, d).

The lumen of these tubules is in some instances filled with a mass (Fig. 5, i) which has become coagulated in the process of hardening, and in other instances with partly cast-off and degenerated cells (Fig. 5, f, h). These cast-off cells show numerous small vacuoles which in life were filled with fat (Fig. 5, h). The nuclei are often multiple, but mostly double, and stain dark.

These changes are to be considered a fatty degeneration which as a rule has affected the cast-off cells only, and is to be attributed to the disturbance in nutrition.

The stroma of the tumor (Fig. 5, a) consists of a loose connective-tissue, containing numerous scattered round cells, which is very sharply distinguished from the tissue of the capsule.

The vessels of the tumor lie for the most part in its immediate neighborhood, but here and there in the septa irregular cavities lined with endothelium are found which contain blood.

On its convex free surface the tumor is ulcerated to a considerable extent. The tumor seems to have perforated the capsule here and finally to have broken through the conjunctiva. This extra-capsular portion shows advanced degenerative changes. Deep in this part the connective tissue is increased and the tubules show a marked fatty degeneration, and on the surface there is merely a necrotic mass.

The microscopic examination makes it certain that the tumor had its origin within the tarsus, and since it has a glandular structure it must have developed from the Meibomian glands or the acino-tubular glands. The whole character of the growth is in favor of the latter view, since besides the location it has a tubular character in common with the acino-tubular glands. As the glands of Krause are surrounded by a loose connective-tissue with numerous round cells, which separates them from the stroma of the tarsus, so this tumor was surrounded by a capsule. In places the tarsal tissue extended between the tubules, but this condition is also found in the normal glands when large masses are surrounded by the tarsal tissue.

There are, I believe, but two cases in literature which are similar to the one just described, inasmuch as they had an epithelial character and the same point of origin. Fuchs'[1] case has less similarity, since there was a proliferation of tubules of a more carcinomatous character, and a more marked participation of the stroma in the process with the formation of cartilaginous tissue.

The case of Moauro[2] is more analogous, as it was a pure adenoma of a very typical structure.

The tumor, of hazel-nut size, was located on the right lower lid of a man of thirty. It projected on the conjunctival surface, and had caused a slight partial ectropium. The skin and muscle over the tumor were normal, and it had its seat in the tarsus.

The base was composed of a dense connective-tissue mass, whose prolongations surrounded the external surface of the tumor and also divided its substance into lobules. The latter were made up of an acinous glandular tissue, imbedded in a delicate reticular connective-tissue containing lymph corpuscles. The acini were of round or oval form, and opened into 2–3 small ducts, which united at acute angles to form larger ducts. All had a distinct basal membrane and a central lumen; the epithelium was everywhere of a single layer, and in the acini the cells were granular and of conical form, with a basal nucleus; the cells of the ducts were cylindrical.

This was a case of new formation of glandular tissue of the most elementary type, *i. e.*, retaining almost all the characteristics of the normal gland, and herein lies its only point of difference from the case reported above, in which there was a proliferation of the epithelial layer, though only within the limits of the acini. The connective-tissue framework preserved its physiological character in all the cases.

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[1] Fuchs, *Arch. f. Ophth.*, vol. xxiv., 2, p. 121.

[2] Moauro, "Adenoma delle glandule tarso-conguintivali di Ciaccio," *Revista internaz. di Med. e Chir.*, vol. iv., No. 4, p. 209.

III.—ADENOMA OF THE MODIFIED SWEAT-GLANDS (OF MOLL) OF THE LID MARGIN.

Antonia S., æt. forty-two, was received in Professor Arlt's clinic May 3, 1880.

The patient first noticed the tumor twenty-five years before. For six years the tumor had interfered with vision. A few weeks before her admission she stuck a thorn into the tumor, but no fluid escaped.

In the inner half of the left upper lid is a rough tumor 27 *mm* long, 23 *mm* broad, and 20 *mm* thick (Fig. 6). It is covered for the most part with the thin skin of the lid, and posteriorly also for a slight distance with conjunctiva. The canaliculus is still to be found on the margin between cutis and mucous membrane near the inner end of the tumor. The tumor is partly of hard consistence, partly soft and translucent as if it contained fluid, but at no point is there fluctuation. The outer half of the upper lid, the lower lid, and the ball are normal.

The tumor was extirpated May 4th without much hemorrhage.

A small piece of the tumor was examined microscopically. It shows a poorly-developed connective-tissue capsule, from which a thick septum passes into the tumor, dividing it into two parts. Both parts have an exquisite glandular structure, yet in one the connective-tissue is much more marked, making the appearance less typical.

The smaller part, which contains only so much connective-tissue as is necessary for the support of the epithelial elements, shows in its central portion an irregular cystic cavity, from which pass in all directions branched tubules with blind rounded ends —a single large dendritic gland of tubular character (Fig. 7).

The inner surface of the cavity and tubules is lined with a double layer of cells (Fig. 8).

The cells of the outer layer (Fig. 8, a) are smaller and more closely arranged than those of the inner layer; they are low, cubical, or cylindrical cells, with darkly-stained nuclei, oval in a direction perpendicular to the wall.

The inner layer is composed exclusively of cylindrical cells (Fig. 8, i) of varying height, but all larger than the corresponding cells of the outer layer. Their nuclei are also larger, more or less oval, and stain less darkly.

The connective-tissue stroma (Fig. 8, s) differs in no way from physiological connective-tissue. It consists for the most part of loose connective-tissue fairly rich in nuclei, and containing a few blood-vessels. Those layers which immediately surround the external layer of cells are of denser structure and have fewer nuclei, representing the membrana propria of the glandular tubules. The most careful examination of the preparations failed to reveal any muscular element in connection with the layers, such as are found in the normal glands of Moll, and only a few isolated smooth muscular fibres were found in the septum.

In the larger part the development of the stroma has been much more marked, and here there is also a wide cystic cavity lined with cells, as in the smaller part. Besides the branched tubules running from this cavity, other glandular tubules are seen often cut obliquely or transversely, and apparently in no connection with the central cavity or its prolongations. These tubules differ from those already described both in form and location, and in the structure of their epithelial lining. The centrifugal prolongations and the dendritic branching are wanting, and a number of regularly-arranged transverse sections of glands are seen in a well-developed stroma.

Both layers of cells have suffered in their development; the outer are reduced to a few flat cells, the inner are better preserved but small and cubical, or flat. The stroma has a denser structure, contains only a few spaces but numerous nuclei, which, arranged in parallel groups, show the stroma to be composed of bundles of young connective-tissue, crossing and interlacing in various directions (Fig. 9). At some points are seen indurated masses of connective-tissue, poor in cells, which send out radiating projections, and thus pass over into the ordinary stroma-tissue (Fig. 10). A dense layer of connective-tissue forming a proper wall of the individual tubules was not found in these portions.

We have here again the characteristic structure of adenoma of the type of a tubular gland. This character and the location of the growth justify us in believing that the origin was in the glands of Moll. There are also other analogies, chiefly in the lining of the glandular tubules. In the normal gland of Moll this is double, the inner layer consisting of cylindrical or cubical epithelial cells, and the outer of endothelial cells.

All these conditions are found: a basal membrane, although not distinct; an outer layer of cells which has lost much of its endothelial character, but differs markedly from the epithelium of the tubule; and an inner layer of epithelial cells which is similar to that of the normal gland of Moll. The deviations from the normal type of gland which the structure of the tumor shows are chiefly as follows: the tubules show many branches, while the normal glands consist of a single spiral tubule; and the sheath of smooth muscular fibre which surrounds the normal gland is here wanting. The latter circumstance is not surprising when it is remembered how seldom muscular elements take part in acquired neoplasms.

The case was not, however, one of pure adenoma. A cystic formation was caused by the dilatation of particular parts of the tubule, and the greater portion of the tumor shows a typical proliferation of connective tissue, which gives these parts a more fibrous appearance. In the heading I have called this growth an adenoma, since the adenomatous character is recognizable in all portions of the tumor, even in the regions of a typical connective-tissue proliferation, and since the beginning of the tumor was purely adenomatous, the fibromatous parts being certainly a later secondary formation. One glance at a preparation will show that the smaller apparently younger lobule, with the central cavity not so markedly developed, has a pure adenomatous structure, while the larger apparently older lobule, with the more extensive cystic cavity, has a more excessive development of connective tissue, and the more advanced the latter, the more markedly do the grandular parts show signs of secondary atrophy.

According to Klebs such connective-tissue proliferations occur not only with epithelial neoplasms springing from the skin, but also often with adenomata. They may be referred in part to embryonic conditions, and they also depend in part on inflammatory processes.[1]

Although this question is difficult to decide in a given case, I am in favor of the former explanation, for at those points where this proliferation begins there are no signs of inflammatory processes, and although groups of round cells and polynuclear leucocytes are found here and there, these changes are not so marked as I have found them, for example, in most cases of carcinoma of the lids.

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[1] Klebs "Allgemeine Pathologie," vol. ii., p. 740.

As far as I know, no case altogether similar has been reported.

If it be now asked what practical purpose these descriptions and considerations serve in the matter of diagnosis and prognosis, it must be admitted that the clinical symptoms are not so characteristic that an accurate diagnosis can be made from them alone. In no one of the cases which I have reported was the correct diagnosis made before the microscopic examination.

In general, the adenomata of the lids seem to be characterized by benignity and slow growth; a striking example is Case III., in which the first appearance of the growth was twenty-five years previous to the operation. In the cases of Baldauf and Moauro, the period was four years; Bock merely makes the general statement that the tumor had existed for years. In Case I. it had developed within a year.

Besides these points, the location and the direction of development may be of diagnostic value in some circumstances. The adenoma beginning in the tarsus will have a certain similarity to chalazion, yet a consideration of the duration of the disease, the size of the tumor, and the appearance of the inner surface of the lid where the gray contents of the chalazion are not seen through the conjunctiva, will make the differential diagnosis. Tumors springing from the orbital margin of the tarsus, and prominent on the inner surface chiefly, have probably originated in the glands of Krause. Adenomata of the Meibomian glands develop sometimes more on the conjunctival side, sometimes more on the cutaneous side, and as a rule attain a larger size than the tumors of the glands of Krause.

The chief point in the diagnosis of adenoma of the glands of Moll is the origin of the tumor in the region of the roots of the cilia.

Adenoma of the lid is in most cases only diagnosed by the microscope. Perhaps these lines will direct the attention of ophthalmologists to the diagnosis of these tumors. My observations have shown that adenoma may develop from each sort of gland that the lid contains. Considering the number of these glands, the adenomata springing from them cannot be so rare as has been hitherto supposed.

Explanation of the Figures on Plates IV. and V.

Fig. 1. I.—Adenoid of the Meibomian glands.

Fig. 2.—Section through the periphery of the tumor. Reichert obj. 2.—h, skin — k, capsule — s, septa — e, the solid glandular tubules, separated from the septa by empty spaces.

Fig 3.—From the middle of the tumor. Reichert obj. 8a — s, septum — l, lymph space, at l′ partly lined with endothelial cells — e, pathological glandular cells.

Fig. 4. II.—Adenoma of the glands of Krause.

Fig. 5.—Part of a large glandular tubule. Reichert obj. 8a — a, stroma — b, single layer of epithelium — c, double layer of epithelium — d, epithelial process, cut longitudinally — e, a similar process cut transversely — f–h, cast-off cells — i, detritus and amorphous contents.

Fig. 6.—Adenoma of the glands of Moll.

Fig. 7.—From the periphery of the tumor. Reichert obj. 4. Branched glandular tubule with regular lining of cells.

Fig. 8.—The part marked x in the preceding figure. Reichert obj. 8a — i, inner layer of cells — a, outer layer of cells — b, basal membrane — s, stroma.

Fig. 9.—From the portion with fibromatous structure of recent date. Reichert obj. 8a — s, stroma with numerous nuclei — t, glandular tubule, in which only the inner layer of cells is preserved.

Fig. 10.—From the portion with fibromatous structure of older date. Reichert obj. 8a — s, stroma relatively poor in nuclei — t, atropic glandular tubules.

TWO CASES OF HEMIANOPSIA WITH AGRAPHIA, AMNESIC APHASIA, AND ASSOCIATED SYMPTOMS.

By Dr. JOHN E. WEEKS.

SURGEON TO THE N. Y. EYE AND EAR INFIRMARY.

(With six wood-cuts.)

Mrs. E. S.,[1] age thirty-nine years. Patient has given birth to four children at term, and has had five still births; two of the children are now alive, the others died of non-hereditary diseases. There is no history of syphilis. The family physician can assign no cause for the still births. The confinements were all very difficult, and were in almost all cases assisted with instruments.

On May 24, 1887, Mrs. S. was delivered of a child at full term. The forceps were used, and there was some laceration of the tissues. Recovery was progressing favorably when, on June 2d, a fire occurred near her apartments, alarming her, causing her to leave her bed and to descend to the street. After walking a short distance her vision failed and she stumbled to the flagging, but did not lose consciousness. She was assisted to her apartments and returned to bed. Patient slept but little during the night. No convulsions, no pain in the head. Twelve or eighteen hours after her fall she noticed partial loss of power in the extremities of the left side. This was gradually and completely recovered from. A defect in the vision persisted, and on December 9, 1887, my friend, Dr. S. Hemingway, kindly sent the patient to my office for an examination of her eyes. The patient was then in good health, with nothing abnormal in her mental or physical condition, except slight anæsthesia of the left side. The points of the æsthesiometer were recognized at half the distance on the right side that they were on the left (the affected) side. Electrical reaction of muscles normal. No disturbance of patellar or other reflexes. Examination of the visual field showed left-sided homonymous hemianopsia. The rough test of comparison with my own eye indicating that the dividing line was vertical. The vision was $\frac{20}{30}$ in both eyes. Color perception normal in the seeing halves. The fundi were normal in appearance. December 27th.—Examination of the urine shows a trace of albumen ; no sugar ; sp. gr., 1027.

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[1] Reported before the Section on Neurology, the N. Y. Academy of Medicine, December 14, 1888.

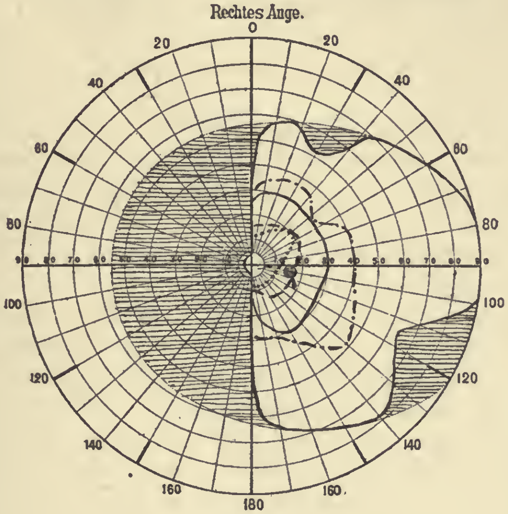
On August 16, 1888, the patient gave birth to a child at full term. Forceps delivery, with slight laceration of vaginal tissues. Recovery progressed favorably up to the afternoon of August 21st, when, while sitting up in bed conversing with her sister, the patient remarked a failure in vision, complained of a strange sensation in her head, laid back on her pillow, and gradually passed into an unconscious condition. There were no convulsions, but during the night the patient muttered unintelligible sounds. She would swallow liquids that were given to her, but the cup had to be placed to her lips. Toward morning when disturbed to take medicine or nourishment she would become excited, weep, and give utterance to disconnected sounds. This condition continued during the day. She rested quietly during the following night.

August 23d.—Patient was quiet during the day ; when spoken to she was unable to understand what was said. Her power of speech was limited to the pronounciation of a few such words as "what" and "yes." Toward evening she could articulate two or three words in succession, as "what then ?" "what is it ?" not, however, as sensible answers to questions. She could not recognize any person or thing by vision alone, but could apparently see objects, and on the morning of August 24th would ask "who 's that ?" or "What is that ?" and when told would indicate by nodding the head and saying "Yes, yes," that she recognized the object. At the request of Dr. S. Hemingway I saw the patient on the afternoon of August 24th. I approached her from the right (seeing) side, but although her attention was fixed on me and she evidently saw me she did not seem to know what or who I was until my name was spoken. She did not recognize familiar objects that were held up to her view until the name of the object was pronounced by some person in her hearing After two or three attempts she could herself pronounce the name. Although the name of the object was recognized she stared at and seemed to study the object as though its shape was strange to her and did not call up the memory of anything that was familiar. I considered this condition to be one of mind-blindness. It gradually passed away. The patient was able to recognize objects in four or five days after the seizure. For about forty-eight hours, beginning about twelve hours after the seizure, Mrs. S. could hear people moving about the room and all accidental sounds, but did not appear to comprehend the meaning of spoken language. The condition is that known as word deafness—the pseudo-aphasia of Broca, and was very transient. The function of the memory centre for spoken language was restored much sooner than that for visual impressions. Tests of the hearing made on the 24th of August showed that to be nearly normal on both sides. There was no paralysis. No anæsthesia of the skin on the left side. No cardiac lesion. Pulse and temperature normal. The interior of the eyes was normal. The left-sided hemianopsia persisted.

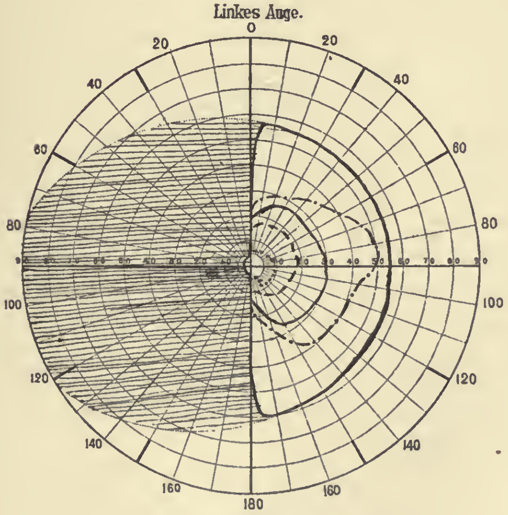
On September 12th the patient was found to be about her rooms performing the lighter domestic duties. She recognized me immediately on my entering the room, but could not remember my name.. Objects were now readily recognized by the visual sense, but were named with difficulty, two or three attempts being usually required before the correct name could be spoken. If the correct name were given her either by voice or in writing, she would immediately recognize it as such, and would repeat it correctly if spoken ; if written she might again mispronounce it. Examples of mispronunciation : A pencil was called "muspel" ; a horse "sorse" ; a slate "slip" ; an envelope "elpolpe" ; cotton "scolly" ; a needle "memadople" ; glass "glopp." The patient reads without more difficulty than that experienced by all left-sided hemianopes. She can neither write, copy, nor draw the form of the simplest object—complete agraphia. Senses of taste and smell normal. She does not remember events that transpired during the first twelve hours of her last seizure. The memory is now about normal for events before and since that time.

September 20th a perimetric measurement of the field of vision showed the dividing line to pass along the perpendicular meridian. At the point of fixation the line described a curve passing into the blind half about three degrees. There are two small defects in the seeing half of the right visual field which are shown in the chart of December 9th.

December 9th.—The patient experiences less difficulty in naming objects correctly. She speaks correctly, but often supplies the place of the proper word by one that is meaningless in the connection. She recognizes the error and endeavors to correct it. She cannot perform even the simplest mathematical problem ;



Right Eye.



Left Eye.

FIG. 1.

cannot count beyond four or five. There is no loss of muscular power.

A second perimetric measurement of the fields of vision was taken (see charts). The defects in the periphery of the temporal half of the field of the right eye indicate, to the author's mind, implication of a small portion of the cortical visual area of the left hemisphere.

In March of 1889, Mrs. S. had another attack consisting of partial loss of muscular power on the *right* side, followed in two or three weeks by *paresis* of the muscles of the tongue and lower part of the face on the *left* side. The hemiplegia passed away in a very short time, but the facial paresis remains. There were no convulsions, no loss of consciousness.

I saw the patient again on May 15, 1891. At that time the vision and fields of vision were as when last recorded. The agraphia remained unchanged, the amnesic aphasia was much less pronounced. Memory for past events was good. She could not count the strokes of the clock above three. Hearing, taste, and smell normal. There was paresis of the muscles in the lower part of the face on the *left* side; the tongue deviated to the left side when protruded. Partial atrophy of the adductor pollicis, flexor brevis pollicis, and interosseus between the first and second metacarpal bones of the *right* hand was observed. Constant chilly sensations in the back and shoulder of the *left* side were complained of. Anæsthesia of both sides was noticed. June 13, 1891.—Perimetric measurement of the visual fields shows but little change. Vision $= \frac{20}{30}+$. No change in the interior of the eyes. Mental and physical condition as on May 15, 1891. The pupils react to light, whether projected on to the right or left sides of the retinæ, and to efforts of accommodation. A careful examination convinces me that the word deafness (sensory aphasia) is not entirely recovered from. An examination of the urine made June 20, 1891, shows a few granular casts; there is no albumen, no sugar. Sp. gr. 1018.

The diagnosis and discussion of this case will follow the report of the second case.

Second Case.—Mr. F., male, age sixty-five years, American, apparently of average intelligence, was admitted to the Alms House Hospital about January 1, 1891, on account of a right-sided hemiplegia. My attention was called to him soon afterward. The following history was obtained: Patient had been a perfectly healthy man, so far as he knew, up to about fifteen years ago, when he experienced paralysis of the right side (leg and arm) ; this passed away in a short time. About ten years after the first attack the patient experienced a second attack of like nature. He claims not to have lost consciousness during either of these attacks, and says that his speech and vision were not materially interfered with. A short time before he was admitted to the Alms House, which was in December, 1890, he experienced a third attack. At this time his vision became somewhat impaired, his speech much affected, and he was unable to write. There was no history of syphilis or of rheumatism.

At the time of my first examination I found a defect of the visual fields consisting of a right-sided homonymous hemianopsia ; the vision was sufficiently good to enable the patient to read ordinary newspaper print readily with either eye.

A subsequent more careful examination showed the vision to be $\frac{20}{30}+$ in both eyes. The dividing line between the seeing and the blind halves of the visual fields was vertical, except at the maculæ where the seeing half extended into the blind half about five degrees. Except possibly a slight paling of the nerves in the halves corresponding to the blind sides, there was no change in the interior of the eyes. The movements of the eyes were normal : pupils of equal size, reacting to the stimulus of light and of accommodation. Speech was interfered with to some extent ; articulation was jerky, and there was evidently difficulty to think of the word desired to express his meaning, occurring in the middle of a sentence. Difficulty to remember the names of familiar objects,—amnesic aphasia,—was marked. If an object, a pencil or a watch, was shown to him, and he was asked its name, he would always fail to give it. However, when he heard the correct name pronounced, he would immediately recognize it as correct. There was no sensory aphasia, no mind blindness, and no alexia, or inability to read. There was complete agraphia. The form of the simplest object, as well as that of the letters of the alphabet, could not be represented with the pen or pencil. The pencil would be grasped as usual, and an extreme mental effort would apparently be made to draw the form ; but meaningless marks only would be the result of his endeavor. Motor aphasia, inability to pronounce only a few short words, as "yes" and "no," developed during the last two weeks of life. The hemiplegia had been recovered from, to the extent of enabling the patient to walk about and to use the arms a little. He experienced difficulty in raising the right arm above the head. There was no deafness. The senses of taste and of smell were normal. There was no hemianæsthesia.

Death from congestion of the lungs, accompanying la grippe, occurred on April 26, 1891. Autopsy by the House Physician.

The lungs were found to be deeply congested, the congestion amounting almost to consolidation. Heart hypertrophied ; liver and spleen apparently normal ; kidneys of the small granular variety. On opening the cranial cavity, it was found to be of normal size. The right hemisphere was of normal size and consistency ; the left hemisphere was much reduced in size. The portion of the cavity not filled by brain substance was occupied by a fluctuating mass, which proved to be a sub-pial cyst, apparently having its origin at the beginning of the fissure of Sylvius. A thin layer of the cortical brain substance from the anterior extremities of the first and second temporal convolutions was raised with the pia forming a portion of the outer wall of the cyst. The pia was also raised from the posterior aspect of the occipital lobe. Shortly after the removal of the brain, the cysts collapsed, and a reddish serum escaped. The brain was placed in alcohol.

After a few days I removed the pia, exposing the convolutions to direct view. The right hemisphere and cerebellum were normal in appearance. The left hemisphere was reduced to about three fifths of the volume of the right. The upper, middle, and anterior two thirds of the lower frontal, the ascending frontal and parietal, except their lower extremities, the supra-marginal, supra-parietal and the uncinate convolutions, as well as the convolutions on the medium surface, back as far as the parieto-occipital fissure, were nearly, if not quite, of normal size. The isle of Reil, posterior third of the lower frontal, lower extremities of the ascending frontal and parietal convolutions, the temporal and occipital lobes and the cuneous, were shrunken and atrophic. The occipital lobe was reduced to about one fifth of its normal size. In the shrunken portions of the brain the diminution in volume appeared to take place largely at the expense of the grey matter. Except in the occipital lobe, where the white matter was also involved, the tissue here was of a reddish hue. There was one focus of softening measuring about 1 *cm* in diameter, located in the white substance of the occipital lobe, immediately beneath the cortical layer. The shrinkage of other parts was unattended with softening. There were no evidences of hemorrhage into any part. The left crus cerebri was much reduced in size. On examining the arteries at the base of the brain, atheromatous plaques were found occurring at frequent intervals in the basilar artery. At the termination of this artery there was a dilatation, apparently a beginning aneurism. The lumen of the posterior communicating arteries was not interfered with. The posterior cerebral artery on the left side was much reduced in size in the portion extending from near its origin to a distance of about one inch. Cross section shows the lumen to be abolished at some points. The artery here resembles a cylinder of connective tissue ; beyond, the lumen of this artery and its branches are almost normal in size. The same state of

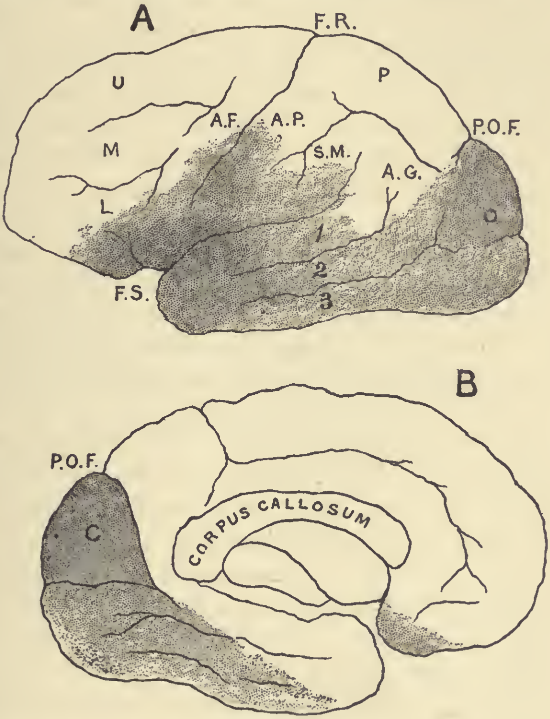


FIG. 2.

Mr. F.—A. External surface. Affected part shaded.
B. Internal surface.

affairs was present in the middle cerebral of the left side, except that a very small opening apparently existed throughout the entire consolidated portion.

Microscopical examination of these vessels shows fibrous thickening of the intima occurring between the elastic lamina and the endothelium. In the posterior cerebral artery this fibrous layer fills the lumen of the vessel. The elastic lamina is seen near the surface of the vessel. The endothelium is crowded at the centre surrounding a very small canal, the contracted lumen. Spaces in this new-formed fibrous mass occur, which are filled with what appears to be fat crystals. The term arterio-fibro-sclerosis has been applied to this condition. It probably originates from some form of endo-arteritis. A complete and probably permanent closure of the lumen of the posterior cerebral occurred at the time of the production of the hemianopsia. The obstruction to the flow of blood through the middle cerebral was evidently not complete, although the lumen of the vessel was greatly reduced. The areas involved are shown in the accompanying figure.

The symptoms manifested in this case, briefly enumerated, are as follows: 1. Homonymous right-sided hemianopsia—left occipital lobe. 2. Amnesic aphasia—isle of Reil. 3. Agraphia—isle of Reil and posterior part of lower frontal convolution. 4. Partial loss of power in the right side, in the upper extremity particularly; motor area of the left side greatest in the middle third of the ascending frontal and parietal convolutions.

The *first* case, briefly summarized, presents the following conditions:

A. Due to lesions on the right side: 1. Homonymous left-sided hemianopsia—occipital lobe. 2. Paresis of the muscles of the lower part of the face, and the tongue on the left side—lower third of ascending frontal and parietal convolutions. 3. Anæsthesia (partial) of the left side; affection of the motor area. 4. The transcient hemiplegia due to temporary derangement of the motor area.

B. From lesions on the left side: 1. Word deafness—posterior third of first temporal convolution. 2. Mind blindness—region of the angular gyrus. 3. Defects in the visual field of the right eye—anterior part of visual area, occipital lobe. 4. Amnesic aphasia—isle of Reil and vicinity. 5. Agraphia—isle of Reil and posterior third of lower frontal convolution. 6. Right-sided hemiplegia—motor area. 7. Anæsthesia of right side—motor area. The permanent defects, from lesions in the *right* hemisphere, are hemianopsia, paresis of the lower part of the face and tongue, anæsthesia. From lesions in the *left* hemisphere, small defect in the visual field, amnesic aphasia, agraphia, anæsthesia.

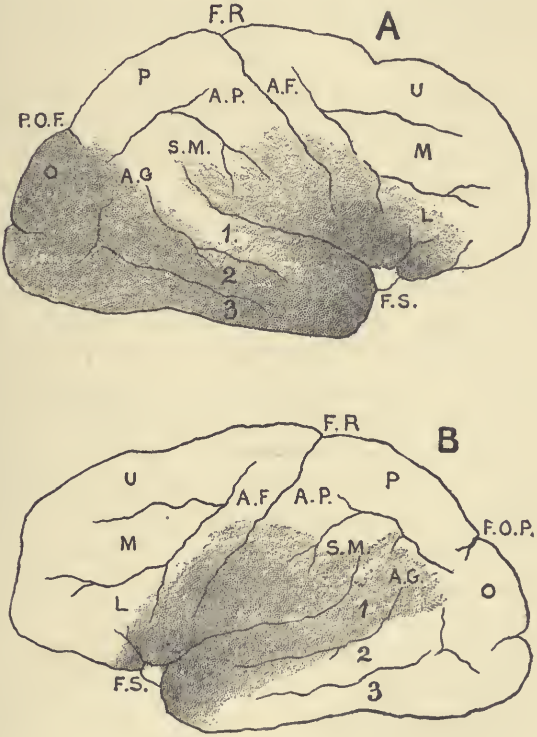


FIG. 3.
Areas probably affected, shaded.
A. Mrs. S.—Right hemisphere. B. Mrs. S.—Left hemisphere.

In the light of reported observations of Wernicke, Charcot, Bernard, Gowers, and others, as well as that afforded by the autopsy in the second case reported in this article, it is safe to infer that the disease of the vessel walls is the prime factor in producing the symptoms in the first case. The middle and posterior cerebrals are probably the seat of an endoarteritis, which has produced a narrowing of the lumen of the vessels and a condition favorable to the formation of thrombi. The first attack was apparently due to an embolism of the posterior cerebral artery on the right side, the embolism originating from her puerperal condition. It is, however, more probable that it was due to the formation of a thrombus, which caused complete occlusion of that vessel. The slow development of a left-sided hemiplegia, such as occurred with the first attack, would favor this hypothesis, as the occurrence of an embolism in the middle cerebral would produce a sudden not a slowly developing loss of power in the opposite side. The occurrence of two of the attacks during the puerperal state is also in favor of this hypothesis, since the puerperal condition disposes to thrombotic processes. There is also in the development of the facial paresis and the extensive anæsthesia, evidence of the slow advancement of the brain affection, which disease of the arterial walls would best explain. In case No. 2 the establishment of collateral circulation through the anastomoses with the anterior cerebral artery appears to have aided in restoring the functions of a portion of the motor area. Either collateral circulation or the re-establishment of the lumen of the middle cerebral, in whole or in part, has served to accomplish the same work in the first case.

The theory of Kussmaul, that agraphia and aphasia occur only as a result of lesions in the left side in right-handed persons, is admirably supported by the first case, as in this case we have a lesion of the area on the right side corresponding to the area for speech and written language on the left side. The lesion on the right side occurred first, but produced no disturbances of speech. The second case is also in confirmation of the theory of Kussmaul.[1]

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[1] The brain described in the second case is preserved in the collection of the N. Y. Eye and Ear Infirmary.

ON TRAUMATIC DIALYSIS (DETACHMENT AT THE ORA SERRATA) AND SECONDARY INVOLUTION OF THE RETINA.

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(With one cut and Fig. 14 on Plate V. of vol. xxii., German edition.)

Translated by Casey A. Wood, of Chicago.

THE retina is firmly attached to the underlying tissues in two localities only—at the papilla and at the *ora serrata*. Throughout the remainder of its entire area it is quite loosely applied to the choroid beneath. In these situations the occurrence of retinal detachment with or without tearing of the tissues, is a common clinical and anatomical observation. On the other hand, it is very rarely observed in the afore-mentioned places where its subretinal attachments are firm and intimate.

Cases of total separation of the membrane from the papilla are quite uncommon. Pagenstecher and Genth[1] have pictured two such instances in their atlas (plate xxviii., Fig. 4 and 5). In both cases this detachment was the result of connective tissue contractions within the retinal "umbrella." In the same way, secondary detachment in the region of the *ora serrata* may also occur.

Sometimes, as Beer points out, it may follow operative interference in glaucomatous (tumor) and staphylomatous conditions. Sometimes choroidal hemorrhages, as well as subretinal effusions, when these forces are not neutralized by a corresponding counter-pressure, result in separation of the retina from the *ora serrata.* In Professor Pagenstecher's collection are preparations of two sarcomata of the choroid which illustrate this. In each case an iridectomy had been made to relieve the glaucomatous tension, but a choroidal bleeding occurred, the retina presented and eventually healed in the wound.

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[1] "Atlas der pathalog. Anatomie des Augapfels," Wiesbaden.

The accident is not, however, always produced by the *vis a tergo;* it may follow directly from an external injury. In support of this contention, I offer the following anatomical report, two clinical histories, and the accompanying illustrations, all of which, with the exception of the history of the second case, I have obtained from Professor Pagenstecher, who has kindly authorized me to publish them.

N. N., æt. thirty-five, ten days before coming to the hospital received a thrust from a cow's horn in the left eye. Severe pain, hemorrhage, and complete blindness immediately followed.

Present condition. Left eye somewhat smaller than normal and much congested. Stretching from the sclero-corneal junction 10.5 *mm* outwards is a slightly depressed, pigmented scar. Iris prolapsed in angles of wound. Cloudy cornea and anterior chamber nearly filled with blood. Total blindness. Right eye normal.

Enucleation of left eye, which was hardened in Müller's fluid and examined nine weeks later. Horizontal section of the bulb into an upper and lower half. Blood-stained fluid, which quite filled both vitreous and anterior chamber, escaped. Antero-posterior diameter, 2.25 *cm;* transverse diameter, 2.37 *cm.* Central portion of cornea very thin, but towards its periphery, especially in the region of wound, noticeably thickened. The wound is closed, closure being effected mostly by superimposed conjunctival tissue. Its margins are slightly turned in; the corneal portion somewhat more than the scleral, so that the latter is overlapped by the former.

The edges are not in immediate contact, but are separated from one another by the intervening iris, which, although prolapsed into the wound, still retains its connection with the ciliary body. Apart from this, and in the lower half of the bulb, is to be seen a torn irregular segment of the iris. This is about 8 *mm* long, and is pushed backward over the *corpus ciliare.* In the superior half of the globe the iris is entirely wanting. Only in the neighborhood of the wound can the individual ciliary processes be plainly seen and distinguished from one another. The remaining and much larger portion of them is concealed below by closely adherent blood extravasation, and above by a delicate, fine-fibred hazy tissue, which clothes the ciliary processes like a membrane, and extends as a tangent in an oblique direction to the upper angle of the wound into which it is healed. There is no trace of lens or vitreous. *Pars ciliaris retinæ* almost entirely absent, except at a small area, 7.5 *mm* wide, which almost exactly corresponds to the locality of the wound. Nearly the whole circumference of the retina is detached from the *ora*, and in places is rolled up inwards. Only in isolated patches, especially where the *pars ciliaris retinæ* is still to be seen, does the retina retain its connection with the *ora serrata*. Here and there the detachment extends in folds to the equator, otherwise it lies along the choroid. There are no hemorrhagic extravasations in the retina ; indeed, with the exception of some thickening at the yellow spot and in the macular region, it presents a normal appearance. Microscopically, thin transverse sections show the retina to be cloudy throughout—due to a molecular fine-celled infiltration.

The changes in and about the macula lutea affect by preference the external and intermediate granular layers. It is an œdematous affection of these parts with thickening of Müller's fibres, where also the molecular cloudiness is most pronounced. The choroid is somewhat thicker than normal, under the microscope the pigment layer is in places loosened from its connection with the elastic lamina by a gelatinous exudation.

If we examine more closely this rare form of injury we can very readily arrange the facts as follows : in consequence of the penetrating wound the eyeball has been ruptured at the sclero-corneal junction ; dialysis and partial prolapse of the larger segment of the iris ; collapse of the remaining portion ; complete escape of the lens and vitreous ; in consequence of the latter accident forcible dragging upon the *ora serrata ;* extensive detachment of the *pars ciliaris retinæ* with its pigment layer ; widespread separation of the retina from the *ora serrata* with a rolling in of the former.

These observations will, perhaps, assist in explaining the cases figured in the "Pagenstecher and Genth's Atlas," plate xxviii., 6, where, in all probability, there had been an injury followed by complete dialysis of the retina at the *ora serrata* with subsequent total involution of the former so that eventually the retina took the form of a pedunculated growth arising from the optic disc.

The following histories are of two cases, the one of a partial and the other of an almost complete separation of the retina at the *ora serrata*, as shown by an ophthalmoscopic examination:

P. W., æt. thirty-five, peasant, seen for first time November 21, 1876. Two days previously received a blow with a stick in the right eye, followed by total loss of vision. With the exception of a recent scar over the superciliary ridge and a slight swelling of the upper lid there is nothing worthy of notice in the tissues surrounding the eye itself. There is a faint conjunctival and subconjunctival injection; cornea clear; no wound to be seen. The anterior chamber is more than half filled with blood. Above is a distinct iridodialysis, and in the same region the pupillary border shows an irregular rent. Tension somewhat increased, V = fingers at three to four feet. Admitted. Treatment with Heurteloup's leech and atropine.

Atropine stopped after eight days. Artificial leech used five times at intervals of five days with marked improvement in vision. Floating opacities in vitreous. Eserine daily for eight days decreased tension. Patient discharged December 24th V with $-\frac{1}{24} = \frac{20}{70}$ and Jäger 5.

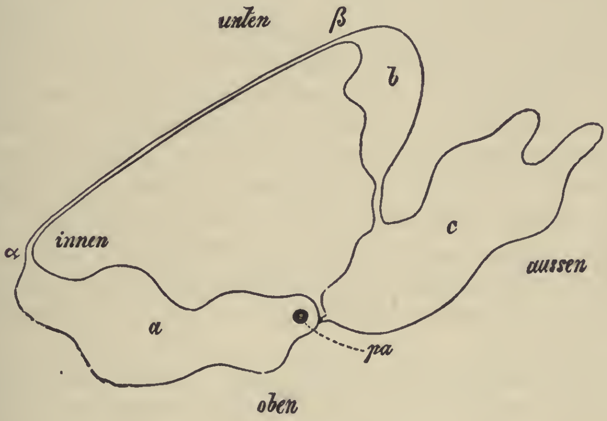
Jan. 20th.—Condition about the same. Eye quiet with V = $\frac{20}{30}$. Mydriasis ad maximum. Transparent lens; vitreous not so cloudy. Fundus normal except at inferior periphery where the retina is hazy, corresponding to a defect in the upper field.

Feb. 7th.—Condition same, with $-\frac{1}{36} \supset$ cyl. $-\frac{1}{48}$ V $= \frac{15}{20} - \frac{15}{15}$.

April 30, 1889.—Patient returned at our request. V = hand-movements at 1 meter; iridodialysis above; tremulous iris; numerous posterior synechiæ; hypermature, shrunken cataract. Visual field everywhere accessible to light. T. normal.

Patient relates that his sight began to fail in the beginning of 1880, and that for the last five or six years it has remained the same. Has never had any pain in the eye. He declined operation.

The foregoing case was undoubtedly one of circumscribed separation of the retina from the *ora serrata*, an essential retino-dialysis. Its limited extent made it unlikely that the retina would float away from the bulbar wall and made it impossible to curl up. Unfortunately the subsequent occurrence of cataract prevented us from making a comparative examination of the fundus after a lapse of thirteen years.



Gertrude W., æt. thirty-eight, on March 20, 1889, while pruning vines was struck in the left eye by a flying twig. This accident was immediately followed by severe pain in the organ and dimness of vision. She came under our care ten days after the injury.

V = doubtful recognition of hand movements in the upper visual field; no p. l. in the lower quadrants; nothing abnormal either by external or by focal examination. Media absolutely clear. When the patient looks straight forwards and slightly downward a peculiar, grayish-white, wavy object is seen whose details are not readily distinguished. The direct examination (best with $+\frac{1}{6}$) shows this to consist of minute folds and coils but no vessels. By the inverted image a striking picture is seen. I regret that it is not possible to represent it by a colored drawing but I trust that my description and the above sketch may help to elucidate the matter.

Not only was the optic disc invisible, but the pupillary area (p. *a*) was hidden by a projecting, easily movable, slightly cloudy, and bladder-like body of a bluish and translucent appearance, whose greatest width was from right to left. At its highest point it measured a disc diameter and a half. Upper and lower borders of the bladder curved sharply backwards. Inwards and somewhat upwards it spread out and covered an area three or four times larger and its surface presented an array of grooves, folds, and processes but no trace of vessels or hemorrhages. Further downwards and inwards (at *a*) the margins of this vesicular body rapidly approach one another, and are continued as a broad choroidal rupture about ¼ d. d. wide, of characteristic appearance, but its course is very unusual, as it stretches outwards and downward obliquely across the periphery of the fundus to (*B*) and joins a smaller vesicle (*b*). As shown in the diagram this smaller body is connected by a pedicle with a larger one (*c*) which is itself divided into two parts by a sort of constricted groove. This bladder-like body (*c*) closely resembles, in size and appearance and other particulars, the one first described (*a*). These are finally joined near the papilla by a delicate fold.

No retinal blood-vessels can anywhere be discovered either in the vesicles or elsewhere, although the choroid in all its details is plainly to be seen.

It may consequently be assumed that the retina has been completely torn away from the *ora serrata*, and has been centrally coiled upon itself. In explanation of the absence of retinal vessels, I would say that when the retina was turned inwards, only the external layers of the membrane were exposed, and that even a slight cloudiness of these sufficed to prevent an ophthalmoscopic view of the blood-vessels.

If we analyze this case we must conclude that the blow from the flying branch sharply compressed the eyeball in its antero-posterior diameter, and as suddenly lengthened its transverse diameter. This action was immediately followed by a stretching of the coats of the bulb. The tough sclerotic and the choroid, loosely attached to the subchoroidal tissue, were able to stand the strain. There was no considerable rupture of the choroidea or counter-rupture of the sclera, but the retinal tissue could not withstand this sudden alternate pressure and stretching; it gave way at its point of attachment to the choroid in the region of the *ora serrata*. Subsequently the contraction of its elastic limiting membrane brought about an involution of the detached retina.

Cases of retinal detachment at the *ora serrata*, the result of penetrating wounds of the bulb or of extensive ulceration of the cornea where lens and vitreous are extended, have been known.[1]

Examples of retinal dialysis with loss of the bulbar contents, are much rarer. I have been able to find two instances only, both of which were, however, accompanied by dislocation of the lens and laceration of the zonula. Fuchs[2] mentioned one of these accidents following an attempt at discission and reclination of a *cataracta arido-siliquata*. The detachment inward and downwards was visible through the coloboma of a preliminary iridectomy. Berger[3] also describes a prolapse of the retina into the anterior chamber with posterior laceration of the lens following a blow from a pitchfork.

On the other hand I do not find a single description of retinal dialysis apart from the dislocation of the crystalline and laceration of the zonula. We may, consequently, regard our first case of partial traumatic dialysis of the retina and iris, and the second, where there was a complete dialysis of the retina, as unique in literature.

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[1] *Vide* "Stellwag's Ophthalmologie," Bd. i., § 713, and Bd. ii., § 552.

[2] *Klin. Monatsb. für Augenheil.*, December, 1877.

[3] These ARCHIVES, xvi., p. 26.

THREE CASES OF COLOBOMA OF THE IRIS.

FROM PROFESSOR FUCHS' KLINIC IN VIENNA.

By Dr. ALFRED POLLAK.

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Translated by Casey A. Wood, of Chicago.

(With figures 11, 12, 13 on Plate V. of vol. xxii., Germ. ed.)

ALTHOUGH coloboma of the uveal tract, directed downwards or downwards and inwards, is comparatively common, this defect is so rare in other situations that the following cases are deserving of special mention.

The first two were very kindly placed at my disposal by Professor Fuchs.

1.—Marie K., æt. fifty. Right eye normal; left affected since birth by a coloboma directed in-and-up. Pupil and coloboma together have the form of a pear (Fig. 11) whose smaller end is directed towards the ciliary border. At the corneal junction a small remnant of the iris is to be seen. The circle of the iris becomes less plainly marked as it gradually approaches the coloboma proper and finally disappears at the root of the defect. The structure of the iris itself is well defined. A cataractous lens prevents a good view of the fundus.

2.—J. S., æt., seventy-six, says she has always had good eyes, that the left has never been subject to an inflammatory attack, and that the defect in this eye has been there her whole lifetime.

There is an almost mature senile cataract in each eye. Although in other respects the right eye seems to be normal, the left iris has an atrophic appearance throughout. At a first glance there appears to be a segment entirely wanting, but a more careful examination reveals an irregular pupil stretching upward, and with the exception of a (at *a*, Fig. 12) small section where it appears to be free it is everywhere joined by a grayish membrane to the anterior capsule. This membrane is also carried upward, exposing only the superior border of the pupil. This latter invests the edge of the gray membrane with a line of dark pigment deposits, which lie on the anterior capsule, in the form of small, tongue-like processes. In the direction upwards from the gray membrane the anterior layers of the iris, as a result of atrophic changes, are represented by scattered remains only, which, for the most part, appear to belong to the posterior layers.

The following is a case of my own:

3.—B. H., aged thirty-two. With the exception of an upward-inward displacement of the pupil the right eye is entirely normal, but the left is small and undeveloped in all its parts. The lower-outer third of the cornea is the seat of a dermoid tumor about the size of half a bean. When the eye is closed the growth occupies a portion of the interpalpebral aperture, and where it touches the upper lid there is a slight defect in the margin of the latter, indicating the site of a small coloboma. The pupils of both eyes are symmetrically eccentric. The temporal half of the left is very black, and the pupil itself reacts promptly to light. The free border of the iris presents to the unaided eye the fine linear and dotted elevations (the so-called "grapeseeds") which are seen in the horse's eye. The intermediate portion of the pupil is filled, or rather replaced, by a very white and compact membrane (Fig. 13). That part of its free margin which presents towards the corneal centre is disposed like a screen behind and almost at right angles to the plane of the iris, and both above and below is slightly curled forward upon itself. It then becomes quickly smaller and as a horizontal process stretches across the iris to the limbus corneæ. Shortly before it reaches the corneal periphery it gives off a delicate branch and in this way isolates a segment of the iris, but appears to have no attachment to the anterior surface of the latter. Although the iris enclosing the normal half of the pupil closely resembles the dark-brown hue of the other eye, and all its details are readily made out, the entire middle half is of a yellow ochre color. It is not noticeably atrophied and exhibits such a uniform appearance that none of its minutiæ can be pictured. The fundus, as far as it can be seen through the small pupil, is normal.

From the capillary margin there extends, towards the left supra-orbital foramen, a zone (about 2 *cm* long and ½ *cm* wide) covered with straight, thick hairs.

In these three instances the iris defect showed itself, as usual, in the left eye.

The first and second cases are of especial interest on account of their direction. Upward colobomata are extremely rare; indeed, with the exception of a passing mention made of one by Ammon, there is in all literature but one other known to me.[1]

The second and third cases also invite our attention to the causation of iris colobomata.

Although Ammon's masterly exposition of the origin of the common (downwards) form is a clear and explicit one, and we know that it is always connected with the fœtal fissure, many theories seek to explain their occurrence in other situations.

Pflüger has expressed[2] the otherwise unsupported opinion that during fœtal life the eye had been rotated 45° outwards, and so remaining has given rise to the outward coloboma. By going a step farther, and supposing an axial excursion of 180°, one might account for even the upward coloboma. This hypothesis will not, however, explain the existence of two colobomata in the same eye, nor of a horizontal iris coloboma, and at right angles to it in the same eye a defect in the choroid.

Makrocki[3] makes an abnormally placed choroidal fissure responsible for these colobomata, nor does Ammon in his work deny the possibility of this. He states that in addition to the physiological fissure which occurs in every eye, a second defect may be present in any situation which remaining open after the closure of the former will give rise to an atypical coloboma.

Abnormalities in the absorption of the pupillary membrane may, according to Plange,[1] have something to do with the origin of colobomata, but no general rule can be laid down on this point, since, as Bock[2] observes, the two conditions very rarely co-exist.

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[1] *Ophthalmic Review*, 1888, p. 310.

[2] These ARCHIVES, xiv., p. 91.

[3] *Archiv für Augenheilkunde*, 1885, p. 73.

Lately the occurrence of intra-uterine inflammation is emphasized as an important etiological factor.

Deutschmann[3] was the first to describe the case of a rabbit's eye affected by a downward coloboma of both iris and choroid. The entire region of the defect was the seat of an active sclero-retino-choroiditis, which the writer thought had produced the coloboma.

Thalberg[4] describes a similar case, involving the entire thickness of the uveal tract in both eyes.

Da Gama Pinto,[5] who does not altogether favor the idea of an inflammatory origin of the defect, thinks the condition of the sclera may have something to do with it.

Schiess-Gemuseus[6] reports two inward-upward colobomata. In one bulb he found along with the coloboma a corneal haze, a shallow anterior chamber, and a pupil displaced upwards, conditions which the writer attributes to the occurrence of a former intra-uterine inflammation.

After a reference to a short description which Talko[7] gives of a horizontal inward coloboma, enclosing a dense white membrane, we shall consider two more instances.

Haase[8] reports an inward-downward coloboma which was covered with a delicate transparent membrane, continuous with the iris tissue, but which did not stretch across the pupillary area. Under the microscope it was shown to be made up of close, felt-like connective tissue, containing cells and nuclei. Corresponding to this, the thinned sclera was covered by an areolar membrane that was indissolubly joined to both choroid and retina. The conditions affecting the membrane in the colobomatous area are analogous to

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[1] *Archiv für Augenheilkunde*, 1890, p. 194.
[2] *Weiner med. Zeitung*, 1888.
[3] *Monatsbl. für Augenheilk.*, 1881.
[4] These ARCHIVES, xiii., p. 253.
[5] *Archiv für Augenheilkunde*, 1888.
[6] *Klin. Monatsbl. für Augenheilkunde*, 1887.
[7] *Monatsbl. für Augenheilk.*, p. 202.
[8] *Archiv für Ophthalmologie*, 1870.

Case 2, where it appears as if a secondary atrophy had followed an inflammatory attack, even so far as in places to have resulted in entire destruction of the iris.

Mittelstädt[1] mentions a horizontal coloboma of the iris, whose floor consisted of bright brownish gray tissue, " such a picture as one often observes after iridectomies where, in consequence of extensive synechiæ, only the superficial layers of the iris are removed by the forceps." In the same eye, also, there was a circular peripheral adhesion of iris and cornea, which the author regards as a result of an old *seclusio pupillæ*, due to an intra-uterine iritis.

Although I do not attempt to explain the inward-upward coloboma in Case 1 by any of the aforementioned hypotheses, I venture to assert that there are many objections to the theory that the atypical defects in the other instances may be due to a fœtal inflammation.

The third case is of interest in another connection.

Van Duyse has attempted in an ingenious monogragh[2] to explain the well-known and not uncommon coincidence of dermoid cysts with coloboma of the lid. He describes a case where a band of cuticle stretched from the sclero-corneal junction backwards to the palpebral margin, then ran parallel to the latter, and was finally inserted into the lid near the inner canthus. This membrane formed a wide bridge with free posterior surface between cornea and lid. Under the microscope it showed the structure of a dermoid tumor.

Bruns[3] makes a similar observation. From the centre of each cornea in an almost full-grown fœtus a delicate band of skin could be traced, which after a short course joined, and as a single process became inserted into the amnion. That coming from the right eye, just before reaching the bulb, divided into two branches, one of which was attached to the cornea and the other to the lower lid.

These are known as Simonart's bands, and are regarded by their discoverer as the outcome of intra-uterine processes, whereby adhesions take place between the inner surface of the amnion and adjacent parts of the body. In this way a cuticular filament may connect the amnion with the eye, and so mechanically hinder the growth of the lid where it touches the latter, while the accompanying dermoid cyst represents the point at which the amnion is attached to the membrane which, in the fœtal state, covers over the eye.

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[1] *Archiv für Augenheilk.*, 1880, p. 426.

[2] Bride dermoide oculo-palpébrale et colobome partiel de la paupière, Gand, 1882.

[3] " Handbuch der praktichen Chirurgie."

Case 3 may then be set down as one of these instances.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE THIRD QUARTER OF THE YEAR 1890.

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WITH THE CO-OPERATION OF

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Sections I.–V. Reviewed by Dr. St. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TEXT-BOOKS, MONOGRAPHS, TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS.

214. Hirschberg, J. Contribution to the history of cataract extraction. *Centralbl. f. Augenhk.*, 1890, p. 198.

215. Kern. Military surgery of the eye. Berlin, 1890, E. S. Mittler & Son.

216. Gordon Norrie (Copenhagen). Georg Heuermann, first German description of cataract extraction (1756), and of Saemisch's operation (1765). Historical notes. *Centralbl. f. Angenhk.*, 1890, p. 261.

217. Utrecht, 1890. Eenondertigste Jaarlysch Verslagvan het Nederlandsch Gasthuis voor hehoeftige en minvermogende Ooglyders gevestigt te Utrecht.

218. Schmidt, E. E. Report on the eye-department of the Marine Hospital, at Sebastopol, for the year 1889. *Med. supplements of the Moeskoi Sborink*, 1890.

219. SCHMIDT, E. E. Observations from the eye-department of the Marine Hospital at Nicolajews. *Ibid.*, Feb., 1890.

220. WIDMARK. Om verkan af de ultravioletta stra, lerna pa ögat. After a lecture at the Berlin Congress, 1890. *Nord. ophth. Tidsskrift.*, vol. iii., No. 2, p. 61.

According to NORRIE (216) George Heuermann was born in 1723, in Oldesloe in Holstein, and died in 1768 in Copenhagen, as professor of surgery. He was the first to practise Daviel's method of extraction in Denmark. In 1765 he described a method of treating the "purulent eye," which closely resembles the one introduced by Saemisch.

In UTRECHT (217) 3,392 new patients were treated, 454 were admitted to the hospital, 367 operations were performed, among them 47 extractions without iridectomy. Snellen says that in infectious ulceration of the cornea tincture of iodine is the most powerful antiseptic. After previous instillation of cocaine the ulcer is brushed with tincture of iodine, and washed off with glycerine. In symblepharon, Snellen transplants a flap with a pedicle from the lid. The pedunculated flap is drawn through an opening, like a buttonhole, and attached with sutures to the inner surface of the lid. WESTHOFF.

SCHMIDT (218) calls attention to the large number of eye patients (as high as 27.8 per cent.) in the navy of the Black Sea. Affections of the conjunctiva are particularly numerous, especially folliculosis and trachoma. The former he considers the lighter forms or early stage of the latter, which supports, therefore, the view of those who maintain that they are one and the same disease. The frequency of trachoma, the existence of which disease debars recruits from admission to the navy, Schmidt explains by assuming that, on account of differences of opinion, some recruits with folliculosis are admitted as being free from trachoma, and thus spread the disease. Trachoma is also introduced from outside. HIRSCHMANN.

(1) In discussing keratitis punctata, SCHMIDT (219) describes two cases in which numerous punctate dots appeared in the tissue of the cornea, in general syphilis, without implication of the uveal tract. In the second case, pannus trachomatosus was superadded. (2) Report of a case of intraocular neoplasm. The left eye of the patient, aged thirty, is atrophic ; where formerly the cornea was, a piece of iron projects from the interior of the eye, which, however, resists extraction. The eye was said to have been

injured seven years previously, and to have run out. No foreign body was observed at the time. Eight months later a piece of iron began to project from the eye without causing pain, but could not be extracted, and did not annoy the patient. Only lately severe pain began to be felt in this eye. After enucleation it was found that the posterior end of the piece of iron, which was 22 *mm* long, was enclosed in a long tumor 7 *mm* thick, round, the size of a pea, firmly attached to the papilla, and composed in its greater part of true bone, with bone corpuscles, and, in its anterior part, of connective tissue, with particles of bone scattered through it. From the choroid the bony tumor is separated by connective tissue. There is no trace of the retina. Schmidt thinks that this bony neoplasm sprang from the retina, and ascribes its formation to the irritation produced by the foreign body. HIRSCHMANN.

WIDMARK (220) gives a brief account of his experiments, showing the effect of the ultraviolet rays upon the anterior media of the rabbit's eye. He also reports that in three of the twelve experiments he found an opacity of the lens ; this seems to him of importance in explaining some cases of cataract from a stroke of lightning, lightning being very rich in ultraviolet rays.

SCHIÖTZ.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

221. CHAUVEL. Un cas de malformation congénitale des deux yeux. *Rec. d'opht.*, 1890, p. 395.

CHAUVEL (221) describes an unusual arrest of development of both eyes. They were of almost normal size and very sensitive to light. T. somewhat diminished. There was horizontal and vertical nystagmus. The corneas were of an elliptical shape The point of the ellipse was directed downward, and here there was an opacity in the cornea. Exactly behind this point the pupil was situated, which in one eye had a diameter of ½ *mm*, in the other 1 *mm*. There was also an opacity at the posterior pole of the lens. Atrophy of the choroid around the papilla with coloboma. $R\ V = L\ \frac{1}{200}$. MARCKWORT.

III.—INSTRUMENTS AND REMEDIES.

222. BEDOIN. De l'emploi des pulvérisations dans le traitement des ophthalmiés. *Bull. de thérap.*, 1890, p. 502.

223. BLANC. Notes d'impressions sur l'ocalistique Américaine. *La clinique*, 1890, p. 401.

224. COHN, HERMANN. The photographic rhombohedron camera especially for taking reflected images. *Centralbl. f. Avgenhk.*, 1890, p. 205.

225. COQUE. Note sur le décentrage des verres de lunettes. *Rev. génér d'opht.*, 1890, No. 6, p. 251.

226. DARIER. Appareil destinié a remplacer la boite des verres. *Ann. d'ocul.*, vol. civ., p. 39.

227. FISCHER, E. A. Modification of Bowman's probe. *Centralbl. f. Augenhk.*, 1890, p. 203.

228. GALLERMAERTS. De l'emploi de la pyoktanine dans la clinique du Dr. Coppez. *Ann. d'ocul.*, vol. civ., p. 55.

229. GUAITA. La pioctanine. Note cliniche. *Ann. di ottalm.*, vol. xix., 2, p. 185.

230. JACKSON, E. A unit of strength and system for numbering prisms. *Ophth. Rev.*, vol. ix., p. 169.

231. JAIS L. Sur les effets produits par l'excentration des verres de lunettes sphériques. Tables. *Arch. d'opht.*, vol. x., No. 5, p. 441.

232. KUBLY, F. Aniline colors in eye diseases. *Wjestnik Ophth.*, 1890, Nos. 4 and 5.

233. LAINATI E DENTI. Sull' impiego dei colori di anilina quali antisettici in oculistica. *Gazz. d'oppit.*, 1890, No. 45.

234. NOGNÈS. De la valeur antiseptique des couleurs d'aniline et de leur emploi en ophtalmologie. *Rec. d'opht.*, August, 1890, p. 438, and *La méd. moderne*, 1890, No. 35, p. 665.

235. NOVELLI. L'olio di anilina in terapia oculare. *Bull. d'ocul.*, vol. xii., p. 15.

236. PEDRAZZOLI. I colori di anilina come antisettici; loro impiego nella pratica, per il Prof. Stilling. Considerazioni e traduz. *Ann. d'ottalm.*, vol. xix., No. 2, p. 123.

237. STILLING. Les couleurs d'aniline comme antiseptique. *Rev. gén. d'opht.*, 1890, No. 4, p. 145.

238. STILLING. Sur l'emploi des couleurs d'aniline. *Rev. gén. d'opht.*, 1890, No. 6, p. 241.

239. STILLING, J. Aniline colors as antiseptics and their application in practice. Second communication. Strassburg, Trübner, 1890.

240. TSCHERNING. Une nouvelle méthode pour mesurer les

rayons de courbure du cristallin. *Soc. franç. d'opht.*, May, 1890. (Tscherning has constructed an instrument for measuring the curvature of the lens, and gives a description of it).

241. VALUDE et AIGNAL. De la valeur antiseptique des couleurs d'aniline. *Arch. d'opht.*, 1890, vol. x., 5, p. 435.

BLANC (223) travelled through the United States for the purpose of study, and praises the instruments used by the oculists of that country. The iris forceps especially he found much smaller and lighter. He also describes at length the squeezing out of the trachoma granules with a forceps constructed for that purpose by Noyes. The operation is done in narcosis, and must be repeated two or three times. MARCKWORT.

COQUE (225) has calculated the prismatic effect obtained by decentring spherical glasses. In biconvex and biconcave glasses the prismatic effect is equal to the decentration (in centimetres) multiplied by the number of the glass (in dioptries) and the number 1.114. In planoconvex and planoconcave glasses the number 0.557 must be substituted for 1.114. MARCKWORT.

DARIER (226) has constructed an apparatus intended to replace the test-case. It consists of two wooden frames which slide upon each other, the one containing 6, the other 7 glasses. The one frame contains: 0, + 1, + 2, + 3, — 3, — 2, — 1, the other, + 0.5, + 7, + 14, — 21, — 14, — 7. The combinations possible are easily found. MARCKWORT.

FISCHER (227) proposes the introduction of caustics simultaneously with Bowman's probes into the lachrymal canal, a plan tried in 1824 by Reisinger with Scarpa's nail. He caused longitudinal furrows to be made, thereby hoping to increase the effectiveness of the probe. Such a furrow can easily be made at the lateral side. It is filled with an astringent or antiseptic substance in form of a powder, and the probe is then introduced. The powder gradually dissolves in the liquid of the lachrymal canal and acts more thoroughly upon the mucous membrane than substances introduced by syringing. The optician Maag, in Dortmund, has such probes in stock.

GALLEMAERTS (228) publishes a number of cases in which pyoktanin was used, in some with good, in others with bad results. Gallemaerts himself says that his observations are not sufficient to permit judgment to be passed on the merits of pyoktanin. Nuel says the same (*Ann. d'ocul.*, vol. civ., p. 58) as to his experiments with the drug. Nuel failed to obtain any improvement in sympathetic disease, but saw good results in conj. blennorrhoica with ulceration of the cornea in an adult.

MARCKWORT.

GUAITA (229), who has used solutions of pyoktanin and the pencils in trachoma, blennorrhœa, hypopyon-keratitis, panophthalmitis and purulent dacyocystitis, speaks unfavorably of the new remedy. DANTONE.

JACKSON (230) reviews the advantages and disadvantages of the various methods proposed for numbering prisms. While all agree that the number of a prism should bear some simple constant relation to its refractive power, the unit of strength and the position of the prism in which the refractive power should be estimated are still *sub judice*. The position of minimum deviation is most easily determined, while the position of refraction all at one surface simplifies calculation. For the unit of strength, the degree has the advantage of being the unit of angular measurement in general use, but it would necessitate a complete change in the present system, and create a need for fractional numbers.

WERNER.

JAIS (231) has prepared three tables, showing in tenths of millimetres the decentration of the glass necessary for every degree of ametropia and insufficiency of the internal recti muscles. The tables are calculated for base-lines of 50, 64, and 75 *mm*.

v. MITTELSTÄDT.

KUBLY (232) tried the yellow and violet pyoktanin (Merck's) in solutions of 1:2,000, 1:1,000, 1:500, and, in a few cases, 1:100; also the pencils, in 500 patients on the lids, in eczema, hordeohum, abscess, blepharitis ciliaris, herpes, injuries, combustion, in acute and chronic phlyctenular, trachomatous, blennorrhoic, and traumatic conjunctivitis; in trachomatous, phlyctenular, parenchymatous, and traumatic keratitis, in corneal ulcers; in corneal ulcers with hypopyon; in various forms of iritis; in iridochoroiditis after typhoid fever; in sympathetic, disseminate, and areolar iridochoroiditis; finally in blennorrhoic dacryocystitis. The drug was well borne in all cases. In none did he see any benefit follow its use. Pyoktanin acts indifferently in all these affections. HIRSCHMANN.

LAINATI and DENTI (233), who tried the blue pyoktanin, are not yet prepared to give a definite opinion as to the effect of the remedy, but only say that it is well borne and harmless. In some undoubtedly infectious diseases it failed to act; in others it did not surpass in any way the antiseptics now in use in ophthalmology. In only one case they obtained an astonishingly quick result, a purulent dacryocystitis being cured in three days by injections of a 2 per-cent. solution. DANTONE.

NOGNÈS (234) publishes therapeutical experiments with aniline colors, conducted at the clinic of Panas. According to Nognès, the eye will readily bear solutions of 1:2,000 to 1:1,000. The latter causes neither pain nor inflammation. In all purulent affections of the conjunctiva, and in all cases of inflammation of the cornea, these substances seem to act as good antiseptics. In trachoma they seem to be less effective. MARCKWORT.

PEDRAZZOLI (236), who has translated Stilling's publications, has himself frequently used aniline-violet in a solution of 1:1,000, and finds that it is not superior to the remedies now in use in acute conjunctivitis, trachoma, and phlyctenulæ. In chronic conjunctivitis and hypopyon-keratitis it is somewhat more effective; and it is excellent in the later stages of interstitial keratitis, while in the beginning of this disease it is not well borne. He anticipates a great future for the aniline colors. DANTONE.

STILLING (237) has experimented with the aniline colors in regard to their antiseptic action, and highly recommends them for asepsis and antisepsis. Of the different colors, methyl violet was used most frequently. His experiments were: 1, botanical and bacteriological; 2, on animals; and 3, therapeutic, on human beings. Methyl violet in a solution of 1:30,000 is said to arrest the development of the bacteria of suppuration. Stilling claims to have obtained very good therapeutic results in corneal ulcers, in hypopyon-keratitis, and even in parenchymatous keratitis, in serous iritis, disseminate choroiditis, and sympathetic ophthalmia. He also claims good results in general surgery. In many cases he saw the pupil dilate in human beings after instillations of methyl violet, without limitation of the accommodation.

MARCKWORT.

In a second paper, STILLING (238) discusses the method of using the aniline colors as antiseptics. He uses them—1, in substance; 2, in form of a larger or smaller pencil; 3, as a powder (1:1,000 to 1:50); 4, as a salve (1:50 to 1:10, in blepharitis ciliaris, eczema, etc.), and in solution (1:1,000 to 1:100).

MARCKWORT.

STILLING'S (239) second communication on aniline colors is divided into a botanical part, prepared by Dr. Wortmann, and a physiologico-medical part. He claims that they possess to a high degree the power of checking the development and also of killing the bacteria and their spores. As to the manner of this action, the bacteria seem to agree in all essential points with the cells of the higher plants examined by Pfeiffer in this direction. Methyl violet also checks the development of the spores of mould, completely preventing their sprouting. He claims the following physiological advantages for pyoktanin: 1, its perfect harmlessness, so that it does not matter whether weak or strong concentrations or the pure substance is used; 2, pyoktanin does not coagulate albumen, which cannot be said of any other known antiseptic; 3, its power of diffusion is extremely great. If, notwithstanding these properties, it should be impossible to obtain any practical results with it, this can lie alone in the fact that the remedy cannot always be brought to the spot where the pathogenic micro-organisms are found. (For a detailed report, see the original.)

VALUDE and VIGNAL (241) conclude from a series of experiments on the action of violet and yellow pyoktanin on streptococcus pyog. and staphylococcus aur., and upon a mixture of bacteria in bouillon, that these substances are very weak antiseptics. Two kinds of methyl violet and one auranine were about equally efficient. Pyoktanin in a solution of 1:5,000 had no effect whatever in ordinary catarrhs of the conjunctiva and in blennorrhœa. In a case of serpent corneal ulcer, which would not heal under any other treatment, there was a surprisingly quick improvement after pyoktanin. A valuable quality of the aniline colors, however, is the ease with which they penetrate into the tissues. They may prove useful in infectious corneal ulcers, when applied in concentrated form. v. WITTELSTADT.

IV.—ANATOMY.

242. BIELONSOFF, ALEXEY. Nervi vasorum. Diss. with twenty-two colored plates (Russian). *Rev.* in *Klin. Monatsbl. f. A.*, vol. xxviii., p. 334.

243. BOUCHERON. Nerfs de l'hémisphère antérieur de l'œil. Nerfs ciliaires superficiels. Nerfs ciliaires externes. Nerfs tendino-scléroticaux. Nerfs cornéens et conjonctivaux. Nerfs de l'espace pectiné. Compt. rend. de la soc. de biol., Paris, 1890. Masson.

244. Cirincione. Sulla struttura delle vie lagrimali dell' uomo. *Riforma med.*, vol. vi., 1890.

245. Delbrück, Anton. Contribution to the knowledge of the decussation of the nerve fibres in the chiasma. *Arch. f. Psych. u. Nervenkrankh.*, vol. xxi., p. 747.

246. Nuel, J. C., and Cornil, F. De l'endothélium de la chambre anterieure de l'œil, particulièrement celui de la cornée. *Arch. d'opht.*, vol. x., 5, p. 309.

Boucheron (242) observed that after complete optico-ciliary neurotomy, the cornea does not become completely insensible. He explains this by saying that only the centre of the cornea is supplied by the deep ciliary nerves, while the periphery is supplied by "superficial or anterior ciliary nerves," which cannot be cut in this operation. These superficial ciliary nerves come from, (1) the orbital nerve at the base of the orbit, (2) the sensory nerves of the muscles, (3) the sensory intraorbital nerves, (4) the nerves of the "espace pectiné," and (5) they form anastomoses with the deep ciliary nerves. Marckwort.

On the basis of careful histological examinations of the structure of the lachrymal passages, Cirincione (244) describes the following tissues, which he claims have thus far not been recorded in literature: (1) in the caruncle a small acinous gland, the duct of which lies at the apex between two large sebaceous glands; (2) in the conjunctiva beside the caruncle small tubular glands, formed by indentation of the conjunctiva and its epithelium; (3) in the lachrymal canals, groups of acinous glands, which, however, are not constantly found; (4) in the lachrymal sac mucous glands, tubular and two kinds of acinous glands; (5) lymph-nodules in the upper part of the nasolachrymal canal, as found elsewhere in the human body. Dantone.

Delbrück (245) had the opportunity to examine the chiasma of a person, aged 70, suffering from dementia, in which complete degeneration of the left, and partial of the right, optic nerve was accidentally found at the autopsy. No ophthalmoscopic examination was made. "The patient could at least find his way without difficulty." The optic nerves and chiasma were divided into cross and frontal sections, and stained, according to Weigert. The author decidedly believes in the partial decussation in the chiasma. He also thinks that he can "safely assert the following: The uncrossed fibres are united in the nerve in thick bundles, which run in a solid mass mainly on the lateral side of the nerve.

In the chiasma, however, and, still more, in the tractus, they gradually mix more and more with the crossed fibres, yet do not spread over the whole cross section of the tractus, but leave free at first the lower inner quadrant, and later a gradually narrowing zone at the whole lower edge of the tractus." It may also be supposed that the uncrossed fascicle lying on the lateral side of the nerve divides in two in the region of the entrance of the arteria centralis retinæ, maintaining the lateral position in a general way, but separating a little upwards and downwards, and thus reaching the globe.

NUEL and CORNIL (246) describe in detail the structure of the endothelium of the anterior chamber. The pigeon and siskin are best adapted for these investigations. But also in mammals and in man the structure of the endothelium is the same in general. In order to preserve the normal conditions of the very delicate endothelium, a solution of osmium was injected into the anterior chamber immediately after the death of the animal and the escape of the aqueous humor, and the enucleated eyeball was placed in the same solution. The protoplasm of the hexagonal endothelial cells consists of very fine fibres, united in bundles and forming with those of the neighboring cells a delicate network. At the point of intersection the nuclei lie, the anterior and posterior surface of which is also covered with the network. They show a special relation to a structureless membrane in contact with the aqueous in the living, to which they adhere when the latter is removed. Numerous illustrations aid in setting forth the details, which cannot be entered into without the former. The authors then describe the cadaveric changes of the endothelium, also those following injury, injection of water, and electrical irritation. Practically it is worthy of note that only a 4 per-cent. solution of boric acid and a weak one of chloride of sodium do not attack the endothelium, while solutions of mercurial salts, on the other hand, destroy all the cells. The regeneration takes place from the iris angle. On the endothelium of the anterior surface of the iris, the structure of which has not yet been sufficiently studied, the authors found in the rabbit numerous stomata, but almost exclusively near the pupillary margin and the ciliary part, very much like the issues and openings described by Fuchs on the anterior surface of the human iris, to which they correspond.

v. MITTLESTÄDT.

V.—PHYSIOLOGY.

247. Arcoleo. Osservazioni esperimentali sugli elementi contrattili della retina negli animali a sangue freddo. *Ann. di ottalm.*, vol. xix., 3 and 4, p. 251.

248. Aubert. The inner language and its relations to the perceptions of the senses and to the movements. *Zeitschr. f. Psychol. u. Physiol. der Sinnesorgane*, vol. i., p. 53.

249. Aubebt. The binocular perimicroscope. *Arch. f. d. ges. Physiol.*, vol. xlvii.

250. Basevi, Vittorio. Fisiologia dei centri innervatori dell' iride. *Ann. di ottalm.*, vol. xix., 2, p. 144.

251. Basevi, Vittorio. De la vision stéréoscopique dans ses rapports avec l'accommodation et les couleurs. *Ann. d'ocul.*, vol. ciii., p. 222.

252. Charpentier. Recherches sur la persistance des impressions rétiniennes et sur les excitations lumineuses de courte durée (suite). *Arch. d'opht.*, vol. x., No. 4, p. 340.

253. Exner, Sig. The disappearance of the after-images in movements of the eyes. *Zeitschr. f. Psych. u. Physiol. d. Sinnesorgane*, vol. i., 1, p. 47.

254. Green, J. L. On colour blindness. *Brit. Med. Jour.*, 1890, p. 234.

255. Helmholtz, H. The disturbance in the perception of the smallest differences of illumination by the proper light of the retina. *Ibid.*, p. 5.

256. Hering, E. A contribution to the knowledge of simultaneous contrast. *Ibid.*, p. 18.

257. Imbert. De l'état de l'accommodation de l'œil pendant les observations au microscope. Paris, 1889.

258. Lipps, Th. On a false projection of the after-images. *Ibid.*, p. 60.

259. Nicati. Notes pratiques. Manière de déterminer plus précisement la position de l'image ophtalmoscopique renversée, en mesurant le degré de la myopie.

260. Querenghi. Contributo clinico alla fisiologia del ganglio ottalmico. *Ann. di ottalm.*, vol. xix., 2, p. 113.

261. Querenghi. Altro caso clinico a contributo della fisiologia del ganglio ottalmico. *Ann. di ottalm.*, vol. xix., 3 and 4, p. 265.

Arcoleo's (247) experiments on frogs and toads on the influence of the nerve-centre on the contractile elements of the retina have shown that after removal of the brain the reaction of the retinal epithelium and the outer portions of the rods and cones to light, electricity, pressure, and heat goes on the same as before the removal of the brain. Other experiments upon the same animals as to the trophic action of the optic nerve upon the retina showed that division of the nerve has no effect upon nutrition, but that division of the ciliary nerves in a short time causes the most diverse disturbances in the choroid and in all the layers of the retina. Dantone.

According to Basevi (250) who made numerous experiments on dogs, cats, rabbits, and especially on pigeons, to determine the seat of the centres governing the movements of the iris, the centre for the sphincter lies in the posterior wall of the third ventricle in front of the corpora quadrigemina and in the medulla oblongata ; a secondary centre lies in the ciliary ganglion. The centre governing the dilatation of the pupil is found between the lower part of the last cervical vertebra and the first two dorsal vertebræ, and at the posterior part of the anterior pair of the corpora quadrigemina and at the posterior pair ; a second centre lies in Gasser's ganglion. The author found no difference between mammals and birds in regard to these centres. Dantone.

Basevi (251) made the peculiar observation that when different colors placed upon the same black background, and therefore in the same plane, are observed, they seem to be at different distances. At a distance of 5 metres yellow seemed to be about 1.5 *cm* and red about 0.9 *cm* nearer than blue. On a colored background these apparent differences changed, but also in accordance with strict laws. Basevi's observations are worthy of the attention of painters for the production of perspective effects. Marckwort.

Exner (253) opposes the explanation of Fick, that the disappearance of the after-images on movements of the eyes is due to the recovery of the retina produced by muscular action, and adheres to his previously expressed opinion, according to which the disappearance of the after-images on movements of the eyes must be accepted as a special case of the general rule, according to which all subjective phenomena disappear on movements of the eyes.

Green (254) mentions a case of yellow-blindness, caused by observation of the sun through a telescope, the dark glass having been displaced for a moment. (No examination with the ophthalmoscope.) WERNER.

According to IMBERT (257) accommodation is scarcely at all called into play in using the microscope, from which he draws the unjustifiable conclusion that the eyes are not more strained thereby than ordinarily. MARCKWORT.

Instead of determining the shortest distance at which the inverted image is still clearly recognized in estimating myopia, NICATI (259) approaches the eye under examination until the inverted image completely disappears and the pupil appears of an even red. The examining eye is then at the focus of the eye examined, and the distance between the two gives the degree of myopia. v. MITTELSTÄDT.

In a young man, in whom a retrobulbar hemorrhage had probably taken place after an injury, QUERENGHI (260) observed amblyopia of the eye in question (discoloration of the papilla), mydriasis, which did not yield even to the strongest light, complete paresis of the accommodation and diminished sensibility of the cornea. The tactile sense of the cornea and the reflex contraction of the pupil on convergence and illumination of the healthy eye remained. The author therefore thinks that as there were no other symptoms of paralysis, the hemorrhage had mainly affected the optic nerve and the ciliary ganglion, and that the following conclusions may be drawn in regard to the physiology of the latter; the accommodation, the pupillary reflex upon illumination of the same eye and the sensibility of the cornea to pain are dependent upon the ciliary ganglion, while the reflex pupillary movement on convergence and on the action of light upon the other eye and the tactile sense of the cornea are directly transmitted from the central organs, with exclusion of the ciliary ganglion and probably through the long ciliary nerves.

DANTONE.

QUERENGHI (161) finds the conclusions just stated confirmed by a second clinical case. The injury was a much more extensive one, and the hemorrhage into the orbit had also caused a weakness of the optic nerve, but the ciliary ganglion had suffered less than in the first case: there was no hyperæsthesia of the cornea, the accommodation and pupillary reaction were decidedly diminished but not abolished, while the reflex movements on convergence and illumination of the healthy eye were the same as in the first case. DANTONE.

Sections VI.–XI. Reviewed by PROF. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

262. BULL. Remarques sur l'examen ophtalmométrique et optométrique d'un certain nombre d'yeux astigmates. *Soc. franç. d'opht.*, May 8, 1891.

263. CHIBRET. Astigmatisme selon et contre la règle. *Arch. d'opht.*, vol. x., 4, p. 357.

264. GIFFORD, H. The needless prescription of spectacles. *N. Y. Med. Record*, July 5, 1890.

265. GULLSTRAND, A. Bidrag til Astigmatismens Teori. *Nord. med. Arkir*, vol. xxii., 2.

266. GULLSTRAND, A. An brännlinier rid Astigmatism. *Nord. ophth. Tidsskr.*, vol. iii., 1, p. 1.

267. HOTZ, F. C. A simple and reliable astigmometer. *Jour. Amer. Med. Asso.*, Sept. 20, 1890.

268. MARTIN, G. Amblyopie astigmatique. *Ann. d'ocul.*, vol. ciii., p. 229. *Soc. franç. d'opht.*, May 8, 1890.

269. NULL. Une curiosité de l'œil myope. *Ann. d'ocul.*, vol. civ., p. 43.

270. SMITH, PRIESTLEY. The causes, prevention, and treatment of myopia. *Ophth. Rev.*, vol. ix., p. 254.

271. STILLING, J. The growth of the orbit and its relation to refraction. *Arch. f. Augenhk.*, vol. xxii., p. 47.

272. TSCHERNING, M. De l'influence de l'aberration de sphéricité sur la refraction de l'œil. *Arch. d'opht.*, vol. ix., 5, p. 445.

BULL (262) discusses the difference in the results of the subjective examination and the ophthalmometric determination, and explains it partly by the rotation of the lens about its vertical axis assumed by Tscherning. According to Bull the hypothesis of an astigmatic spasm of accommodation is not justified.

MARCKWORT.

CHIBRET (263) finds a greater number of astigmatic patients since he has used sciascopy. Among 569 patients 6.5 per cent. were astigmatic, 5.2 per-cent. being according to rule, and 1.3 per-cent. against it. The latter form was *not* discovered with the ophthalmometer, but only by sciascopy combined with the subjective test. In using the former the astigmatism is not measured in the line of vision but 1 *mm* to the temporal side of it, thereby appearing less or being absent entirely. The subjective test may, however, agree with the result found with the ophthalmometer, as a part of the As. can be corrected by astigmatic accommodation. This part sometimes reaches 2 D. Sciascopy alone can determine in such cases the total degree of astigmatism. As. against the rule occurs in about one fifth of the whole number of cases, is frequently not attended by any As. discoverable with the ophthalmometer, and can only be determined by sciascopy. It sometimes reaches 2 D. The correction found subjectively lies between that obtained with the ophthalmometer and that found by sciascopy, and approaches the former in As. according to the rule, and the latter in As. against the rule. In practising sciascopy, after neutralizing any ametropia the examiner may possess, he advises the determination and correction of the refraction of one meridian, and then to compare, at a distance of 80 *cm*, not the *movements* of the shadows, but the *differences in their brightness*. He claims that a difference of 0.25 D can easily be recognized and that the accuracy of this test is far superior to that of the examination in the erect image. v. MITTELSTÄDT.

GIFFORD (264) protests against the routine prescription of spectacles for low forms of ametropia, particularly in children.

BURNETT.

In developing the theory of astigmatism GULLSTRAND (265, 266) has admitted into the equations of a pencil of light the infinitely small quantities of another degree, and without using the approximation of Matthiesen or the theory of Sturm he has arrived at results materially differing from those thus far accepted. They could be verified by experiments and applied to pencils of light of finite size. He found that the ordinary astigmatic pencil of light need not have any focal line whatever.

There are certainly two thinnest cross-sections at both focal points, so that when for instance the principal meridians are horizontal and vertical, the pencil of light is thinnest at the one focal point in a transverse, at the other in a vertical direction; the cross-sections, however, are generally not linear, but of a certain width, which can be calculated for a given pencil of rays; in a general way they are neither perpendicular to the principal ray of the pencil, on the contrary they may form with it any kind of an angle. Three forms may be set up. The first form has two straight focal lines, which at the two focal points are perdicular to the principal ray. The second form has a straight focal line, which usually forms an acute angle with the principal ray. This is the most important form, which occurs in the human eye, as he has also shown by experiments. The third general form for the astigmatic pencil of light has no straight focal line, and need not have any focal line at all.

SCHIÖTZ.

HOTZ'S (267) apparatus is but a modification of Donder's round point of light. He has three points of light close together and arranged so that they can be rotated on a disc. When the three are elongated and point in the same direction, that marks one of the astigmatic meridians. It is very similar to Purvis' apparatus devised some years ago. BURNETT.

MARTIN (268) endeavors to explain those cases of astigmatism, in which perfect correction cannot be obtained, by assuming an astigmatic amblyopia, which he thinks develops when the retina from earliest childhood receives only blurred images of lines of a certain direction. Martin says: "When the correction (by astigmatic accommodation) is insufficient, the image is diffuse in one direction, and in this direction a persistent amblyopia appears, which I propose to call astigmatic, in order to designate its manner of development." Armaignac, Leroy, and Javal express themselves rather doubtfully in regard to Martin's explanation. MARCKWORT.

In his latest publication MARTIN tries to disprove the objections raised by Leroy and Armaignac at the *Soc. franç. d'opht.* (May, 1890), and endeavors to show that astigmatism may actually cause amblyopia; as the images formed upon the retina are indistinct in one axis on account of the astigmatism, the retina becomes less sensitive in this axis, therefore amblyopic, as in cases of congenital cataract which have not been operated upon at an early age. In subjectively determining the best correcting-glass Martin chooses that one with which those three lines are best seen upon the large plate of Snellen bearing the triplicate radii, which previously appeared the most indistinct. Sometimes even with the best correction all the lines are not equally black.

MARCKWORT.

NUEL (269) endeavors to prove that the judgment, which we form after retinal irritation as to the direction and distance of the objects seen, may change when the position of the retina changes. According to Nuel the retina is displaced so much in increasing myopia and staphyloma that retinal lines formerly vertical become inclined. If judgment did not also change, such myopes would see vertical objects inclined and vice-versa.

MARCKWORT.

PRIESTLEY SMITH (270) objects to the view that myopia is the result of redundant growth and indication of a higher state of intellectual development, and considers that it is mainly an expression of a weakness of the tunics. When the condition is stationary he would give the same glasses for reading and distance. WERNER.

STILLING (271) thinks that the higher the percentage of myopes, the lower the average index of the orbit, the more myopes there are, the more the average indices of myopes and emmetropes will increase, and the lower will be the average index of the latter ; the more emmetropes there are, however, the higher the average index will be. The difference in the indices of emmetropia and myopia will be the greater, the larger the number of emmetropes, and the higher the average index for emmetropia will be.

TSCHERNING (272) finds in the spherical aberration of the cornea the cause why, in the higher degrees of As., the ophthalmometer marks it too low, because the refraction considerably increases towards the periphery in the meridian of greatest curvature, much more than in the meridian of least curvature. The ophthalmometer does not measure the radius of curvature at the apex of the cornea, but that of a circle 1 *mm* distant from it ; this radius is larger. The As. of the cornea can be corrected and even over-corrected by the spherical aberration of the lens, but this cannot neutralize the increase of As. of the cornea towards the periphery without the lens itself being astigmatic. The spherical aberration of the lens explains without the aid of other theories the increase or decrease of refraction observed after atropine, also the increase of astigmatism. From this the practical conclusion can be drawn that too great a value should not be attached to the measurements found in mydriasis, and that a spherical or astigmatic spasm of accommodation should never be assumed in a case of decrease of refraction during mydriasis, or that there is astigmatism without a test with a diaphragm. In conclusion he advises, in opposition to Chibret, not to attach absolute confidence to the results of an objective examination. The subjective test must always be the one which finally guides us. v. MITTELSTÄDT.

VII.—LIDS.

273. Gillet de Grandmont. Greffe de peau de grenouille appliquée à la restauration des paupières. *Soc. franç d'opht.*, July, 1890.

274. Pooley, T. R. Operation for ectropion of the lower lid by the sliding-flap method. *Amer. Jour. Ophth.*, Sept., 1890.

275. Rumschewitsch, K. The onkology of the lids. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 387.

Gillet de Grandmont (273) showed at the Paris Ophthalmological Society a girl, aged thirteen, in whom he had used the skin of a frog's belly for a blepharoplasty of both lids of the right eye. After detaching the edges of the lids from the orbital margin, to which they had become adherent by scars, and after performing the blepharoraphy, he covered the two raw surfaces, after previously drying them, with small pieces of skin (3 to 4 sq. *mm*) from the skin of a frog's belly. The result was very good. After Despagnet and Valude had remarked in the discussion that the same results could be obtained with pedunculated flaps from the neighborhood, Darier said that, if that were the case, the transplantation of the skin of an animal was to be preferred.
Marckwort.

Pooley (274) was entirely successful in relieving an ectropion of the lower lid, the result of an operation for removal of a malignant growth by means of a sliding flap drawn from the temple and cheek. There was no suppuration of the flap whatever, and the resulting deformity is very slight. Burnett.

Rumschewitsch (275) describes a case of lymphangioma, and one of epithelioma of the lids. He also gives the results of the examination of a granuloma of the lid, an adenoma of Krause's glands, the sebaceous glands and the modified sudorific glands of the edge of the lid, a dermoid of the lower lid, a papilloma of the edge, and an adenoma of Meibom's glands.

VIII.—LACHRYMAL APPARATUS.

276. Cirincione. Tracoma dei canaliculi lacrimali. *Giorn. dell' assoc. dei naturalissi e medici*, vol. i., 4.

277. Dufour. Oreillons des glandes lacrymales. *Rev. méd. de Suisse Romande*, 1890, p. 490.

278. Norrie, Gordon. Parotitis epidemica begyndt i faarekirflerne. *Nord. ophth. Tidsskr.*, vol. iii., 1, p. 30.

279. Sgrosso. Contributo alla dacrioadenite acuta. *Ann. di ottalm.*, vol. xix., 2, p. 159.

Cirincione (276) had the opportunity to examine the eyes after death of three persons affected with trachoma, and found that the canaliculi also took part in the trachomatous process, their walls showing the same changes as the conjunctiva. In a fourth corpse the whole mucous membrane of the lachrymal sac was covered with trachoma-granules, which were also found numerously in the canaliculi, but only in small numbers in the fornix ; the development had therefore begun in the lachrymal sac. The author mentions that thus far in only one case, described by de Vincentiis, trachoma was demonstrated in the lachrymal conducting apparatus with the microscope.

Dantone.

A girl aged eleven presented herself to Gordon Norrie (278) with slight catarrhal conjunctivitis, and the outer part of the upper lids was slightly swollen. The following day the swelling of the lids was more marked, but mainly confined to the region of the lachrymal glands. The next day mumps. The lachrymal and parotid glands were of the same consistence.

Schiötz.

Sgrosso (279) reports on two tumors at the outer canthus, which were incised and proved to be genuine abscesses of the lachrymal gland. Dantone.

IX.—MUSCLES AND NERVES.

280. Browne, E. A note on the conjugate movements of squinting eyes. *Ophth. Rev.*, vol. x.; p. 222.

281. Casper. Combined paresis of the ocular muscles by direct injury. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 451.

282. Darkewitsch, L. O. Recurrent paralysis of the oculomotor nerve. Moscow, Sept., 1890. Reprint.

283. Eissen, W. Three cases of functional disturbances of the movements of the eyes. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 427.

284. Fuchs. A rare case of paralysis of the facial and abducent nerves. *Wiener klin. Wochenschr.*, 1890, No. 9.

285. Mocosow, L. A case of functional torticollis due to congenital concomitant squint. *Wratsch*, 1890, No. 33.

286. NORRIE, GORDON. Noyle sjaldene Tilfcelde af Nystagmus. *Nord. ophth. Tidsskr.*, vol. iii., 1, p. 24.

287. REICH. Co-ordinate spasm of the superior recti muscles. *Wjestnik Ophth.*, 1890, Nos. 4 and 5.

288. SCHNELLER. Contributions to the theory of squint. *von Graefe's Arch. f. Ophth.*, vol. xxxiv., 3, p. 138.

289. TAYLOR, S. T. Miner's mystagmus and the safety lamp. *Brit. Med. Jour.*, Aug., 1890, p. 480.

290. VALUDE. Le strabisme névropathique. Étude de pathogénie. *Arch. d'opht.*, vol. x., 4, p. 326.

291. VIALET, N. De la cure du strabisme dans ses rapports avec l'acuité visuelle de l'œil devié. *Arch. a'opht.*, vol. x., 4, p. 289.

BROWNE (280) states that in cases of convergent strabismus the centre for conjugate movements acts as in normal eyes, but that when once the limit of internal movement has been reached by one eye, the external rectus of the fellow-eye is able to exert an independent and supplementary action. WERNER.

After a blow in the region of the left inner canthus CASPAR (281) saw paresis of the left internal rectus and of the trochlearis, which healed under local faradization.

DARKEWITSCH (282) furnishes an analysis of relapsing paralysis of the oculomotor nerve on the basis of a case observed by himself and twenty collected from the literature of the subject. The constant symptoms are: uniocular ptosis, deviation of the eye outward, checked mobility inward, upward, and downward, mydriasis, and paralysis of accommodation, the function of the other eye not being impaired. Inconstant symptoms are: anæsthesia or paræsthesia of the upper branch of the fifth nerve, nausea, vomiting, headache. This combination of symptoms and the recurrence of the affection make the diagnosis certain. When the statements of the patient and the symptoms are not reliable, youthful age, constant headache on one side, nausea, and vomiting, the absence of all other symptoms of paralysis and the short duration of the paralysis are in favor of this diagnosis. The cause may be a chronic affection of the nerve on its way at the base of the brain from meningitis, neoplasm, etc. The relapses are probably due to temporary disturbances of circulation.

The prognosis is doubtful. All methods of treatment have thus far been unsuccessful. (After a review in *Wratsch*, No. 35.) HIRSCHMANN.

Eissen (283) describes three cases of functional disturbance of the ocular movements in highly nervous persons.

In one the abducent was affected, in the second the oculomotor, and in the third all the external ocular muscles. In all of them there was concentric limitation of the visual field. As the general health improved the ocular affection disappeared. All three cases were probably due to hysteria.

In a girl of fourteen, who in her third year had suffered a fracture of the skull and now showed paralysis of the left facial, the left abducent, paresis of the acoustic and of the motor portion of the fifth nerve, Fuchs (284) observed a gaping of the left palpebral fissure, which remained open even when the attempt was made to close the eye. There was also marked convergence due to the contraction of the internal rectus.

Mocosow (285) saw a case of functional torticollis due to congenital concomitant squint. The patient's head was turned towards the left shoulder and downwards about 35°, and the left side of the face projected a little more than the right. The left eye had V $\frac{40}{40}$, the right $\frac{28}{40}$. Both E. While the head was in the position just described the irregularity of the position of the eyes was scarcely noticeable. When the head was held straight the right eye at once deviated distinctly upwards and outwards.

Hirschmann.

Gordon Norrie (286) reports on seven cases of nystagmus. In two of them an injury was followed by nystagmus in one eye with rotation of the head to one side, both of which symptoms disappeared in a short time. Schiötz.

A drover, thirty years old, otherwise healthy, but somewhat pale and apathetic, presented himself at Reich's (287) policlinic with a spasmodic sursumduction of both eyes, which returned every ten minutes or even more frequently, and lasted about four or five minutes. During the spasm there appeared under the forcibly raised upper lid only the lower edge of the cornea, which appeared with a slow movement, sometimes more, sometimes less. In the intervals no pathological changes could be found in the eyes. The affection had lasted about ten days, and followed a fever of three days' duration. Injury or headache was denied. After the internal exhibition of bromide and iodide of potassium the attacks rapidly decreased. Reich has found in the literature of the subject only one analogous case of intermittent tetanus of the superior recti muscles, namely, that of Samelsohn. Hirschmann.

Taylor (289) maintains that insufficient light is quite enough to cause nystagmus without any peculiarity of position, just as a dense corneal leucoma may produce it. The reason "holers" are so liable to it is that, (1) the light is worse where they work; (2) "holing" is more exhausting work than any other in coal mines. Werner.

After reporting the clinical history of thirty cases Valude (290) comes to the conclusion that the ordinary concomitant strabismus does not depend on ametropia alone and that the explanation of Donders perhaps does not even fit the majority of cases, but that a neuropathic disposition is an important, sometimes the principal factor in the development of strabismus. There are cases in which ametropia alone, in others the nervous affection, in still others both combined, are the cause. The treatment, accordingly, must vary, and in not a few cases must be directed against the constitutional affection. Sometimes this treatment alone is sufficient to effect a cure. When these principles are neglected the strabismus, though removed by an operation, may return. By neuropathy the author does not necessarily mean nervous degeneration of epileptic, hysterical or neurasthenical persons. Simple nervousness may be sufficient. It can be transmitted to children and cause strabismus. Hysteria may also cause strabismus without ametropia. The latter is the predisposing, the former the incidental cause.

v. Mittelstädt.

Vialet (291) first presents a review of the various diametrically opposed views as to the relation between the amblyopia of the deviating eye and strabismus. Among 150 cases of strabismus in Landolt's clinic, he found 122 with amblyopia. From these he selected 51, which were frequently examined and carefully tested to decide this question. Of these 51, there was convergent strabismus in 45 and divergent in 6 cases. There was no improvement after the operation in only 8 cases, and increase of visual power is therefore the rule. Improvement was mainly observed in convergent strabismus, brief existence and slight amblyopia. In old and high degrees of amblyopia, which the author considers congenital, improvement is very rare. The author believes in amblyopia from disease, and thinks that after the eye has been straightened, the refractive error corrected and the weak eye stimulated to take part in the visual act, the visual power will improve and binocular vision then becomes possible. v. Mittelstädt.

X.—ORBIT AND NEIGHBORING CAVITIES.

292. Eulenburg, A. On astasia-abasia in Basedow's disease. *Neurol. Centralbl.*, 1890, No. 23.

293. Panas. Considérations cliniques sur les abscès des sinus frontaux pouvant simuler des lésions indépendantes de la cavité orbitaire. *Soc. franç. d'opht.*, May 8, 1890.

294. Snell, S. Sinus of left orbit associated with disease of right upper central incisor. *Ophth. Rev.*, vol. ix., p. 193.

In a girl suffering with Basedow's disease, Eulenburg (292) observed astasia and abasia, inability to stand or walk.

In Snell's (294) case, that of a girl æt. 17, a sinus formed at the lower margin of the left orbit as a result of disease of the right upper incisor, which had been fractured by a fall. The sinus closed after its removal. Werner.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

295. Alleman, L. A. W. Galvanism in the treatment of corneal opacities. *Brooklyn Med. Jour.*, Nov. and Dec., 1890.

296. Blessig, E. Report of a case of subjunctival cysticerci. *Wjestnik Ophth.*, 1890, No. 4, p. 321.

297. Braunschweig, P. Contribution to the knowledge of infantile xerosis of the conjunctiva. *Fortschritte d. Med.*, 1890, No. 23.

298. Burnett, Swan M. Racial influence in the etiology of trachoma. *Med. News*, Nov. 22, 1890.

299. Keinig, H. and O. A method of treating trachoma successfully tried in numerous cases. *Deutsche Med. Wochenschr.*, 1890, No. 41.

300. Kollock, Ch. A form of xerosis. *Ophth. Rev.*, vol. ix., p. 249.

301. Magee, R. S. Corneal ulcers, with special reference to their treatment by the galvano-cautery. *Kansas Med. Jour.*, Nov., 1890.

302. Mæurer, C., Jr. Cauterization of the conjunctiva with mercurial precipitate, and the simultaneous internal exhibition of iodide of potassium. *Arch. f. Augenhk.*, vol. xxii., p. 24.

303. Norrie, Gordon. Xerosis of the conjunctiva. *Nord. ophth. Tidsskr.*, vol. iii., 1, p. 31.

304. Norrie, Gordon. Tuberculosis of the conjunctiva. *Nord. ophth. Tidsskr.*, vol. iii., 1, p. 19.

305. Raehlmann, E. On trachoma. *Deutsche Med. Wochenschr.*, 1890, No. 41.

306. Rosmini. Sull' uso del sublimato corrosivo nella terapia oculare e specialmente nella cura dell' ottalmia granulo-tracomatosa. *Boll. d'ocul.*, vol. xii., Nos. 14, 15, 17, 18, 19.

307. Schapringer, A. Inflammation of Tenon's capsule as a result of influenza. *N. Y. Med. Record*, June 14, 1890.

308. Truc. L'ophtalmie granuleuse à Montpellier. *Montpellier Mèd.*, 1890, p. 406.

309. Wheelock, K. K. Tropho-neurotic keratitis. *N. Y. Med. Record*, July 26, 1890.

Alleman's (295) method is to place one pole over the cornea, covered by the lids, and the other on the cheek, just below the lower orbital edge. Sometimes it is placed on the naked cornea, rendered anæsthetic by cocaine. He usually begins with a strength of $\frac{1}{4}$ to $\frac{1}{2}$ milliampère. The histories of ten cases are given. In all cases, even those of many years' standing, improvement was noted, and in some it was very pronounced.

Burnett.

From the observation of five cases of xerosis of the conjunctiva in children, all of whom died of internal affections, Braunschweig (297) concludes that the bacillus of xerosis is not the cause of the constitutional affection. It is an accidental, harmless settler on previously diseased and disintegrating tissue. Xerosis is only a phenomenon secondary to other pathologico-anatomical changes in the body.

Burnett (298) again calls attention to the fact, first brought to notice by him in 1876, that the negro in the United States seems to enjoy an immunity from trachoma. He also instances some facts which seem to show that trachoma may flourish at and above an altitude of 1,000 feet. He considers trachoma a dyscrasic affection and not a mere local disease, and that it should be treated accordingly.

H. and O. Keinig (299) treat trachoma by energetically rubbing the conjunctiva of the lids and fornix with a pledget of cotton, dipped into a sublimate solution (1:2,000), no matter whether it is ulcerous or bleeds. This is repeated once or twice daily. When the granules occupy a spot which is difficult to get at, they crush them with a ciliary forceps. They also wash out the eyes three times daily with a lukewarm sublimate solution (1:1,000). Sometimes after very energetic rubbing there is intense swelling of the lids; then the friction must be discontinued for one or two days. By this method of treatment the trachoma is completely cured in from two to six weeks, and the corneal affections are also healed.

KOLLOCK (300) describes a form of xerosis occurring in negro children, especially in scrofulous and emaciated subjects. The ocular conjunctiva becomes thickened, greenish-yellow in hue, and is often thrown into folds which are capped with silvery scales. The disease does not follow any form of ophthalmia, frequently gives rise to ring-ulcer of the cornea, and hemeralopia is sometimes complained of. It most nearly resembles a type reported by Bitot and Blessig to have occurred in Russia.

WERNER.

MAGEE (301) reports six cases of corneal ulcer in which the galvano-cautery was used with more or less success in stopping the morbid process. He thinks it specially useful in those cases having their origin in mycotic infection.

BURNETT.

In a patient, into whose eye on account of a corneal ulcer he had introduced a salve composed of white mercurial precipitate, oxyde of zinc, and vaseline, and who was also using an iodine salve on account of orchitis, MÆURER (302) saw cauterization of the conjunctiva. Small quantities of iodine were found in the urine.

GORDON NORRIE'S (303) case was that of a child eight months old, which had fallen ill a few days previously. The lids of both eyes were kept half open; the conjunctiva was dry and slightly scaly, the cornea dry and dull. In the left cornea there was an ulcer with a necrotic protruding mass. After a severe, probably cerebral, disease the child recovered, contrary to expectation. The right eye became entirely normal again, the left was destroyed by panophthalmitis. Compresses dipped in a solution of boracic acid had been constantly applied. SCHIÖTZ.

RAEHLMANN (305) calls trachoma all follicular affections of the conjunctiva. The anatomical and clinical peculiarities of trachoma arise from the changes in the follicles. The follicular conjunctivitis of other authors is trachoma. The formation of follicles takes place either rapidly or slowly. In both forms they spread in two different ways, they are found either superficially or in the depths. When the superficial follicles develop in an acute manner, the accompanying inflammation is of a catarrhal nature. The mucous membrane is thrown into folds and wrinkles, at the apex of which the follicles are found ; the lower lid is particularly affected, especially the fornix. They are more rarely found upon the conjunctiva of the upper lid. In the chronic form there is frequently no sign of inflammatory irritation. The mucous membrane is swollen, the surface wrinkled, the follicles are arranged in rows, but are larger, pale, and diaphanous. Healing may take place both in the acute and chronic forms spontaneously, after a longer or shorter period, without leaving a trace. By many they are called follicular conjunctivitis, and are considered different from trachoma. Raehlmann cannot support this view, as he has seen the most severe kinds of trachoma develop from this condition. In the severe forms there is an extensive proliferation of follicles both on the surface and in the depth of the tissue. The fate of the mucous membrane depends upon the changes in the follicles. It is rough and nodular, slightly or not at all folded, rather tensely swollen. The follicles are crowded together beside, behind, and over one another in the conjunctiva of the lids and the retrotarsal folds. In consequence of perforation of the follicles, ulcerations form, which in turn lead to the formation of scars. The tarsus is curved, and keratitis pannosa may develop. Trachoma is contagious without doubt, but the discharge probably possesses contagious properties only at times. Transmission through the air is impossible.

ROSMINI (306) discusses the new teaching of the parasitical origin of the granulations (follicular conjunctivitis) and trachoma, and praises sublimate as the principal remedy ; he calls it the king of antiseptics. DANTONE.

SCHAPRINGER (307) gives an account of a suppurative tenonitis associated with a degree of panophthalmitis in a man affected with phthisis which seemed to be due to the grippe, no other cause being found. The patient was still under observation when the report closed. BURNETT.

WHEELOCK (309) reports three cases of keratitis in which there was infiltration and other signs of inflammation, but without pain, photophobia or lachrymation, and with almost complete anæsthesia of the cornea. They were treated by galvanism, which acted pleasantly and favorably in all the cases. He thinks the seat of the disease to be in the Gasserian ganglion, from which the trophic nerves seem to come. Internally he gives strychnine.

BURNETT.

310. OBRASZOW and SERGEJEW. A case of keratitis punctata syphilitica. *Wjestnik Ophth.*, 1890, Nos. 4–5, p. 323.

311. SGROSSO ANTONELLI. Contributo, alla anatomia patologica della cornea negli occhi atrofici. *Ann. di ottalm.*, vol. xix., 2, p. 116.

In OBRASZOW and SERGEJEW's (310) patient, a man aged sixty-two and amaurotic from other causes, numerous small dots developed in the parenchyma of the cornea of both eyes (about 50 in the right, 30 in the left), about 1 *mm* in diameter, white, and lying at different depths, about four months after the primary infection, together with several primary sclerosis of the prepuce, swelling of the lymphatic glands, macular and papular syphilides, destruction of the mucous papillæ of the tonsils, and of the mucous membrane of the mouth and lids ; there was also moderate, deep-seated pericorneal injection. At the centre of the cornea they were crowded closer together and were smaller than at the periphery. Between the dots the cornea is almost perfectly clear. Treatment with injections of salicylate of mercury ($\frac{1}{6}$ to $\frac{1}{3}$ of a grm.) caused all the constitutional symptoms to disappear, while the corneal dots also gradually cleared up, each from its centre towards the periphery. It was now found that also the right iris was infiltrated to a very great extent, and became atrophic as the infiltration disappeared. With the disappearance of the corneal dots the author also observed the formation of small depressions in the cornea (without loss of substance), but not corresponding to the dots. They consider this a case of keratitis parenchymatosa punctata, as described by Mauthner, Raehlman, and Fournier, complicated in the right eye with parenchymatous iritis.

HIRSCHMANN.

SGROSSO and ANTONELLI (311) describe the changes in the epithelium, Bowman's membrane, and the tissue itself of the cornea of four enucleated atrophic eyeballs. The indentations of the membrane, its complete absence in spots, and especially its perforation by delicate offsets of the parenchymatous cells (canalization of the subepithelial tissue according to de Vincentiis) are carefully shown and illustrated with excellent drawings of the microscopical conditions.

DANTONE.

Sections XII.–XXI. Reviewed by Dr. P. SILEX.

XII.—IRIS.

312. Dujardin. Iridectomie par un procédé simplifié. *Jour. des sci. med. de Lille*, 1890, No. 24. p. 554.

313. Van Duyse. Tuberculose oculaire. *Ann. de ocul.*, vol. civ., p. 5.

314. Lange, O. Tumor corp. ciliar., rupt. capsulæ lentis, catar. consec. traumatica. *v. Graefe's Arch. f. Augenhk.*, vol. xxxvi., p. 277.

315. Lembeck, H. On the pathological anatomy of total congenital irideremia. *Inaug. Diss.*, Magdeburg, C. Friese, 1890.

316. Rieke, A. Foreign bodies in the iris. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 375. (A chip of stone in the iris for thirty years with good sight).

317. Seggel. Another case of a chip of steel remaining in the iris for years; removal by iridectomy, good sight. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 291.

318. Terson. Presentation d'une malade opérée depuis sept mois d'une tuberculose de l'iris. *Soc. franç. d'opht.*, May 6, 1890.

319. de Wecker. La tuberculose de l'iris. *Soc. franç. d'opht.*, April 1, 1890.

320. Zirm, E. A case of persistent pupillary membrane adhering to the cornea. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 288.

Dujardin (312) operated in a case of occlusion of the pupil after cataract-extraction in the following manner: below the horizontal corneal diameter he made two small wounds with a Graefe knife by puncture, counter-puncture, and a few sawing movements. Through one wound he introduced the iris forceps, through the other the pince-ciseaux, and then excised in the anterior chamber a central piece of iris, which he removed with forceps. Marckwort.

Lange (314) finds that only four cases of tumor of the ciliary body accompanied with cataract have been published, and carefully describes one observed by himself. The symptoms always present were unequal depth of the anterior chamber, irregular dilatation of the pupil and dislocation of the opaque lens. The development of these cataracts has been explained in various ways: the tumor pressing upon the lens was said to cause a disturbance of nutrition similar to choroiditis, others ascribed it to the detachment of the retina and increased pressure, though this explanation referred rather to the later stages, still others laid it to the mechanical insult. Lange found as the cause a rent in the capsule caused by the pressure of the tumor. The latter consisted of hypertrophic and hyperplastic smooth muscular fibres and spindle-shaped connective-tissue cells, and was therefore a myosarcoma.

TERSON (318) showed a patient from whose iris he had excised a tubercle. V increased after the operation from $\frac{1}{10}$ to $\frac{1}{2}$. In a similar case de Wecker could perform only a partial resection; nevertheless recovery took place. Coppez saw tuberculosis of the iris heal simply under constitutional treatment. Galezowski is opposed to excision; he saw recovery under constitutional treatment combined with repeated paracenteses of the anterior chamber. MARCKWORT.

DE WECKER (319) reports on two patients with tuberculosis of the anterior part of the uveal tract. Both of these eyes had been condemned to enucleation by other oculists. He succeeded in preserving both by appropriate constitutional treatment (arsenic and iodoform combined with a milk-cure). One eye regained $V = \frac{1}{2}$; the other became slightly phthisical. These cases have induced de Wecker to advise an operative procedure in tuberculosis of the eye when the disease is confined to a small spot, but to refuse to operate (therefore not to enucleate) when the affection has spread to the whole anterior part of the uveal tract. While de Wecker assumes that tuberculosis of the eye is generally due to endogenous infection, Chauvel and Parinaud hold the opposite view. Vignes and Parinaud therefore advocate enucleation in case the functions of the eye in question have been destroyed. Despagnet is opposed to iridectomy for excising the tubercle, as it is impossible to know whether the process is confined to the iris. Abadie and Gillet de Grandmont assert that the diagnosis cannot be considered certain until tubercle bacilli have been shown to exist and inoculations have succeeded. In regard to the technique of iridectomy for tuberculous nodules de Wecker states that he first makes a small incision through the cornea above the nodule, and enlarges it downward with the scissors, as the peripheral position of the tubercle makes the usual form of incision impossible. MARCKWORT.

XIII.—CHOROID.

321. Bock, E. Contribution to the knowledge of the neoplasms of the eye. *Centralbl. f. prakt. Augenhk.*, vol. xiv., p. 1.

322. Garnier, R. Contribution to the knowledge of panophthalmitis. *Wjestnik Ophth.*, 1890, Nos. 4 and 5.

323. Guende. Une observation de néoplasme choroidien developpe chez une femme atteinte de cancer du rein. *Rec. d' opht.* 1890, p. 325, and *Soc. d'opht. de Paris*, June 3, 1890.

324. Pincus, O. Case of choroidal sarcoma. *Deutsche med. Wochenschr.*, 1890, p. 791.

325. Sgrosso. Neoformazione di cartilagine nella membrana ciclitica e tessuto neoformato prechoroideale di un occhio atrofico, *Ann. di ottalm.*, vol. xix., 2, p. 178.

326. Trousseau. Iridochorioidite cataméniale. *Soc. d'opht. de Paris*, June 3, 1890.

Bock (321) describes a sarcoma of the choroid in a child aged three, a melano-carcinoma of the limbus, a sarcoma polyposum of the ocular conjunctiva, and a glioma of the retina.

According to Garnier (322) the term "panophthalmitis" belongs to a group of symptoms, a clinical picture, which may be based on various pathological processes and etiological factors. In proof he minutely describes five eyeballs, enucleated on account of panophthalmitis and examined by himself (one after injury, four after cataract extraction). For the details of the pathological changes in the iris, choroid, retina, vitreous, and papilla the original must be consulted. Microbes were present in all, though they showed nothing specific. Some resembled the septicæmic streptococci, others the pus-staphylococci; many varieties of micrococci were found. Cultures did not succeed. In one case the microbes entered by way of a prolapse of iris, in three others remnants of lens-capsule prevented adaptation and healing of the wound, so that the microbes obtained access. The manner in which the vitreous was infiltrated with pus confirms, in the author's opinion, the view of Straube, that the vitreous is divided into concentric, funnel-shaped layers by eight to ten membranes extending from the papilla to the ora serrata. The two layers, the uveal and retinal, genetically different from the iris, may become affected independently of each other and in a different manner. Endovasculitis obliterans occurs both in inflammation of the stroma of the iris and in anterior synechiæ and prolapse of iris. The marked infiltration of the ciliary body with cells is due to the great vascularity. The author never saw proliferation of the muscular elements. The œdema of the papilla can generally be explained by the checked escape of the liquids of the eye. He never found dropsy of the sheath of the optic nerve. HIRSCHMANN.

PINCUS (324) found a melanotic sarcoma in a phthisical eyeball. Graefe has already described such globes affected with sarcomas of the choroid. The eyeballs are painless, the form peculiar, as the axis is small, while the lateral diameter is large. Extraocular masses of tumor lying behind the eyeball sometimes cause a slight degree of exophthalmus.

SGROSSO (325) found several deposits of hyaline tissue in the so-called cyclitic membrane of an exceedingly small atrophic eyeball. DANTONE.

TROUSSEAU (326) describes a peculiar form of iridochoroiditis with hypopyon, which always began in a patient a few days before the menses and disappeared in a few days. This state of affairs lasted for a long time. With the beginning of pregnancy the attacks ceased for a time ; one month before delivery, however, another attack set in ; the next occurred before the second menses. After that they regularly returned. Gorecki, Duboys de Lavigerie, and Despagnet report similar cases during the discussion. MARCKWORT.

XIV.—GLAUCOMA.

327. BIRNBACHER. Contribution to the anatomy of acute glaucoma. Graz, 1890.

328. COLLINS. Iridescent vision in glaucoma. *Ophth. Rev.*, vol. ix., p. 196.

329. FANO. Le glaucome. *Journ. d'ocul. et de chir.*, 1890, p. 151.

330. ROGMAN. De l'hyphéma post-opératoire. *Ann. d'ocul.*, vol. civ., p. 139.

331. STÖLTING. Cure of buphthalmus in two cases. *v. Graefe's Arch. f. Augenhk.*. vol. xxxvi., 3, p. 263.

BIRNBACHER (327) accidentally had an opportunity to examine an eye affected with glaucoma. For details the original must be consulted. It is worthy of note that the vitreous was free from changes. An abnormal arrangement of the column of blood was found in the capillaries, the smaller and medium-sized veins of the choroid, manifesting itself in an increase and marginal position of the leucocytes, the walls of the blood-vessels being intact, and also in an increased albuminous exudation. Chronic pathological proliferations of the endothelium were found in the two upper vortex veins. These latter changes he and Czermak had demonstrated years ago in chronic glaucoma also. As long as the circulation is normal in other respects, no disturbances will probably result. But when other alterations besides supervene, for instance, increased afflux or diminished efflux, compensation may become impossible, and a sudden increase of tension is the result.

From the results of experiments with erythrophleine COLLINS (328) concludes that the colored halos produced by it, and those which occur in glaucoma are due to the same cause, viz.: steaminess of the cornea from superficial epithelial changes. Erythrophleine produces slight anæthesia and steaminess of the cornea with foggy vision and colored halos, but no increase of tension. As in glaucoma, the red ring was outermost, independently of the refraction. The halos were independent of fixation of the light, accommodation, size of pupils, and presence or absence of the lens. WERNER.

FANO (329) discusses the various kinds of glaucoma. In regard to the treatment of simple glaucoma, he says neither iridectomy, nor sclerotomy, nor the production of a so-called filtration-scar cures that form of glaucoma. MARCKWORT.

ROGMAN (330) publishes a very interesting paper, in which he calls attention to the fact that in consequence of hemorrhage into the anterior chamber after an operation, the tension can be markedly increased. He cites three cases in confirmation of this view. In the first two cases the hemorrhage occurred during the operation, and in the third, two days later, the increase of tension always simultaneously with the hyphæma. Repeated punctures had a good effect. He then thoroughly discusses the literature of the subject. MARCKWORT.

STÖLTING (331) performed sclerotomy for buphthalmus on a child seven months, and on another two years old, and considers the cases cured. They were under observation for eighteen months. In one eye the iris prolapsed and a cystoid scar formed. [In such cases Prof. Schweigger has performed iridectomy for many years, as only rarely good results were obtained in adults with sclerotomy. The reviewer has had several such patients under observation for four or five years, but would hesitate to say that they are cured. He will report later as to the results.]

XV.—SYMPATHETIC OPHTHALMIA.

332. Ayres, S. C. Case of sympathetic inflammation after panophthalmitis of the injured eye. *Amer. Journ. Ophth.*, Nov., 1890.

333. Brailey. Discussion on sympathetic ophthalmia. *Brit. Med. Journ.*, 1890, p. 411.

334. Galezowski. De l'ophtalmie sympathique et du moyen de traitement par un debridement circulaire du globe oculaire. *Rec. d'opht.*, July, 1890, p. 138.

335. Teillais. Deux cas d'ophtalmie sympathique. *Soc. Franç. d'opht.*, May 6, 1890.

In Ayres' (332) case there was suppurative panophthalmitis following an injury to the eye from broken glass. The eye atrophied. Four months later the other eye began to be affected with plastic iritis of a slow form. The stump was not sensitive, but was removed. Burnett.

The discussion was opened by Brailey (333), who made the following remarks: The exciting eyes exhibit a plastic uveitis with clusters of cells in the iris, ciliary body and choroid, but the chorio-capillaris and pigment-epithelium generally escape. Sympathy occurs after serous and suppurative disease also, and from non-perforating tumors. Papillitis was the first sign in 10 per cent. of all cases, in 5 per cent. it does not go further, it is generally a serous uveitis with punctate keratitis and + T. It seems to travel through the nerve-sheaths, and then along the vessels or episcleral tissue. The bacillary theory does not explain its non-appearance after suppuration, and in non-perforating tumors. Deutschmann described his well-known experiments on the subject. Story considered that the general septicæmia which occurred in these experiments lessened their value. Werner.

Galezowski (334) believes that the cause of every sympathetic affection must be sought in the ciliary nerves. Instead of enucleation he makes an operation which so detaches the eyeball from its surroundings, that only the connection with the muscles and conjunctiva remains. For this purpose Galezowski enters Tenon's capsule with curved scissors at the four recti muscles. On the nasal side he excises at the same time a piece of the optic nerve. MARCKWORT.

According to TEILLAIS (335) sympathetic ophthalmia may develop in consequence of a slight contusion, without the aid of any infection. Sympathetic ophthalmia of ciliary origin cannot therefore be abandoned. The course of this disease differs materially from that of ophthalmia migrans. Abadie and Boucheron agree with Teillais. In this form of sympathetic ophthalmia enucleation, which is insufficient in ophthalmia migrans, is especially indicated. In these cases Boucheron found neuritis of the ciliary nerve. MARCKWORT.

XVI.—LENS.

336. ASTENGO. Lavaggio della camera anteriore nell' operazione di cataratta. *Boll. d'ocul.*, vol. xii., p. 16.

337. BOURGEOIS. De la Kystectomie dans l'opération de la cataracte. *Rec. d'opht.*, Aug., 1890, p. 456.

338. DUFOUR. De la cataracte secondaire au point de vue opératoire. *Soc. franç d'opht.*, May 6, 1890.

339. FAGE. Infection tardive de l'œil après l'opération de la cataracte. *Ann. d'ocul.*, vol. civ., p. 36.

340. FROTHINGHAM, G. E. A case of static lenticular astigmatism acquired by long-continued use of spectacles having a faulty position. *Jour. Amer. Med. Assoc.*, Oct. 25, 1890.

341. MCHARDY. Treatment of immature senile cataract. *Ophth. Rev.*, vol. ix., p. 254.

342. KALISH, R. The absorption of immature cataract by manipulation conjoined with instillation. *N. Y. Med. Rec.*, Dec. 20, 1890.

343. KERSCHBAUMER, R. Report on 200 cataract extractions. *Arch. f. Augenhk.*, vol. xxii., Nos. 2 and 3, p. 127. This number of these ARCHIVES.

344. KNAPP, H. On extraction of lenses dislocated into the vitreous. *Ibid.*, p. 171.

345. KNAPP, H. Division of the capsule during the corneal section in cataract extraction. *Ibid.*, p. 186.

346. KNAPP, H. Report on a third hundred of cataract extraction without iridectomy. *Ibid.*, p. 191.

347. Ledda, S. Délire chez les opérés de cataracte. *Rec. d'opht.*, 1890, p. 398.

348. Nikoljukin, N. Report on 304 cataract extractions, performed in 1888 and 1889 at the Peter-Paul Hospital. *Wjestnik Ophth.*, July–Oct., 1890.

349. Parinand. Du délire après l'opération de la cataracte. *Soc. franç d'opht.*, May 7, 1890.

350. Pitts, Barton. Cataract extraction. *N. Y. Med. Rec.*, Oct. 18, 1890.

351. Popow, A. Report on 103 cataract extractions. *Wjestnik Ophth.*, July–Oct., 1890.

352. Rolland. De l'opération de la cataracte chez les maladés ambulants et de la responsibilité médicale qu'elle entraine. *Rec. d'opht.*, 1890, p. 216.

353. Schweigger, C. On extraction of unripe cataracts. *Deutsche med. Wochenschr.*, 1890, p. 695.

354. Scimeni. Sulla modificazione della curratura della cornea in seguito ad estrazione di cataratta. Osservazioni ophthalmometriche. *Ann. di ottalm.*, vol. xix., 3, 4, p. 211.

Astengo (336) reports on 16 extractions in which he did not perform iridectomy, but washed out the anterior chamber according to de Wecker. The result was excellent in all of them.

Dantone.

Bourgeois (337) has constructed a new capsule-forceps, with which he removes a piece of the anterior capsule in opening the lens-capsule. After the former has been seized near the centre, the capsule is rotated about its longitudinal axis and then withdrawn. The object therefore is not to tear out a piece of the anterior capsule by traction, but by torsion. Marckwort.

Dufour (338), after discussing the operation of secondary cataract in every direction in his review, comes to the following conclusions: 1. The operation for secondary cataract is justified only when unpleasant consequences can be excluded. 2. It is now possible to completely avoid infection in this operation. 3. The operation is indicated only when V has been reduced by the secondary cataract to less than $\frac{20}{50}$. 4. At least 6 to 7 weeks must elapse between the extraction and the operation of the secondary cataract. 5. The operation is performed with a needle, when there is merely a membrane; with Graefe's Knife, when then there are iritic adhesions; with the pince-ciseaux, when there is a thick, firm (inodulaire) secondary cataract. 6. The needle must be so made that bits of the secondary cataract will not easily adhere to it and be drawn into the corneal wound. De Wecker recommends to divide the capsule according to Green at right angles to the direction of the traction of the iris. In the discussion a great difference of opinion manifested itself as to the advantages of the different instruments. Panas, Wecker, and Suarez de Mendoza prefer the pince-ciseaux; Martin and Motais Graefe's Knife; Galezowski the instrument of Desmarres or a curved needle, and Meyer the small hook. To the question of Lasalle and Dor, as to how the complete secondary cataract should be operated upon, which does not form until a long time after extraction, Dufour answers: by iridocapsulotomy according to Wecker, therefore with the pince-ciseaux.

MARCKWORT.

FAGE (339) publishes two new cases of panophthalmitis in eyes previously operated on for cataract. In the first case in which the affection developed 4 months after the extraction, enucleation was performed. In the second case, in which the extraction had been done 10 years previously, the disease was checked with Saemisch's incision and antiseptic treatment. MARCKWORT.

FROTHINGHAM (340) reports the case of a young man who showed an astigmatism of 0.75, axis vertical, after a long-continued use of glasses of + 3 D for near work, which were so fitted in the frame as to stand at an angle of 23° with the vertical meridian. At a previous examination under prolonged use of atropine he had manifested a H of 0.75 D. BURNETT.

MCHARDY (341) adopts Foerster's method of maturing cataracts, performing preliminary iridectomy at the same time, whenever vision becomes less than $\frac{6}{24}$. It ripens a senile cataract in from eight days to eight weeks. Rupture of the capsule is very uncommon, slight iritis often occurs, and the cortex is not sticky. Panas, Snell, and others approved of the method.

WERNER.

KALISH (342) reports the histories of seventeen cases of lenticular opacities, many of which are figured, which were treated by his method of manipulation, or rubbing of the eye through the lids after instillation of glycerine, boric acid, and rose-water. The opacities were mostly diffuse and of recent date. The paper was discussed (disapprovingly, Ed.) by the ophthalmologists at a meeting of the Medical Society of the County of New York.

BURNETT.

Ledda (347) reports on some more cases of delirium after extraction, and even after the simple instillation of atropine (5 or 6 drops of a solution of 0.05 to 30.0), without an operation or bandage. Ledda recommends chloral, bromide of potassium, and especially hydrobromate of quinine to prevent the delirium.

Marckwort.

Nikoljukin (348) operated on the first eighty-six eyes according to v. Graefe's method (linear, peripheric extraction), and had panophthalmitis in two cases (accidental infection), occlusion of the pupil in four, glaucoma in one (serous iritis), protracted course of healing in five, with good result, however ; incarceration of the iris in five, but without injurious consequences, and thickening of the capsule in four. To prevent injurious consequences, Nikoljukin laid the section entirely in the cornea in the following fifty-five eyes. Among these he had one case of iridocyclitis, terminating favorably ; two of occlusion of the pupil ; one of suppuration, ending also in occlusion of the pupil. In two, sight was poor, on account of thickening of the capsule. To prevent secondary cataract, Nikoljukin endeavored to remove the lens within the capsule in the following thirty-one eyes. He extracted it with the loop. In most of them, prolapse of vitreous took place (being entirely absent in only twelve) ; but he ascribes no special importance to it. In one case, panophthalmitis occurred, in one occlusion of the pupil, in one detachment of the retina, which the author, however, is not inclined to ascribe to the loss of vitreous. In spite of good results, and to prevent the rupture of the capsule and the failure of the extraction within the capsule, Nikoljukin did not remove the lens in this manner in patients of middle age, in increased intraocular pressure (when the eyeball remains tense after the corneal section) and in liquefaction of the vitreous. In consequence he did not have any loss of vitreous in twenty-one among the following sixty-two cases. In thirty-three the extraction was performed without iridectomy (generally in hypermature cataracts). In seventeen he obtained a round pupil, in thirteen it was drawn upward and irregular, in three incarceration took place. The best vision was obtained in cases with a round pupil. Double vision was present : the image belonging to the right eye stood to the right and lower and its upper end tips outwards. When the head is inclined, double vision cannot be produced, even with colored glasses. For his explanation, the original must be consulted. Hirschmann.

Parinaud (349) discusses the causes of delirium, which frequently occurs three days after extraction. Without denying the influence of atropine and alcoholism, he considers the occlusion of the eyes the principal cause. Armaignac holds the same view. Grandclément and Galezowski accuse atropine in particular; Chibret, alcoholism.
Marckwort.

In more than one hundred cases of extraction, one fourth of which were without iridectomy, Pitts (350) has not had a single instance of corneal suppuration, plastic iritis, or panophthalmitis. Two failures in eyes which were diseased before operation, in all the others V ranged from $\frac{20}{90}$ to $\frac{20}{20}$.
Burnett.

Popow (351) had eight losses among his first forty-two operations, which were done under very unfavorable hygienic conditions. Among the next sixty-one, done under more favorable auspices, there were only five losses. The minute statistics are unsuitable for review. In suppuration and blennorrhœa he obtained good results with instillations of a solution of chinin. sulph. gr. viii. ad ℥ j.
Hirschmann.

Rolland (352) discovered that some oculists treat cataract patients like out-door patients. They let them walk or ride home immediately after the operation had been performed in the consulting-room. He severely condemns the procedure, and thinks it so objectionable that it should be punished by law.
Marckwort.

Schweigger (353) thinks that the terms ripe and unripe cataract are improper as regards the question of the operation. The point to be settled is whether the lens is in a condition to be extracted, *i. e.*, whether it can be removed without leaving any larger remnants behind. This is possible when the lens has lost its elasticity, *i. e.*, when it has become flatter and the power of accommodation has disappeared. This happens at about the age of sixty, and every lens causing marked disturbances of vision can therefore be extracted at that time of life, even when considerable portions of it are still clear. At an earlier time, between forty and sixty, the clear portions form a sticky substance, which remains behind after extraction, swells, and frequently destroys the eye by rendering the cornea opaque and causing choroiditis. To render such a patient able to work again, an operation is also advisable, but not until after previous ripening by massage according to Foerster, which may also be done without iridectomy. In young persons division is indicated.

Scimeni (354) has undertaken a second series of ophthalmometric measurements of the cornea after cataract extractions (in which he also took into consideration the periphery) and has found: (1) that the cornea does not lose its ellipsoid form after extraction, and that the meridians passing through the line of vision generally remain elliptical, as before the operation; (2) that the curvature increases in the meridians parallel to the section, that the angle α decreases but remains positive; (3) that the eccentricity of the ellipse diminishes in the meridians perpendicular to the section, so that the curvature of the cornea approaches the sphere, the angle α becomes larger in the positive direction, the angle α' almost zero, while the apex of the ellipse falls below the centre of the meridian. These changes are most marked soon after the operation, and subsequently disappear in many cases. Dantone.

XVII.—VITREOUS.

355. Hircshberg, J. On the formation of blood-vessels in the vitreous. *Centralbl. f. prakt. Augenhk.*, vol. xiv., p. 266.

Hirschberg (355) saw in four patients formation of blood-vessels into the almost clear vitreous, following hemorrhagic inflammation of the retina and optic nerve. He seeks the cause for the latter in diabetes, syphilis, and arterio-sclerosis. The blood-vessels may disappear.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

356. Abadie. Traitement du décollement de la rétine. *Soc. franç. d'opht.*, May 7, 1890.

357. Adamück, E. On detachment of the retina. *Wjestnik Ophth.*, July–Oct., 1890.

358. Bernhardt, P. On disturbances of the visual field and changes in the optic nerve in neurasthenia and hysteria. *Inaug. Diss.*, Zurich, 1890. A. Diggelmann.

359. Bjerrum. Et tilfcelde of hemianopsia partialis. Helbredelse. Localdiagnose mulig. *Nord. ophth. Tidsskr.*, vol. iii., 2, p. 71. Copenhagen, 1890.

360. Dunn, John. A case of reflex amblyopia cured by section of the supraorbital nerve. *N. Y. Med. Jour.*, Aug. 9, 1890.

361. Goldziehr. On a case of endarteritis obliterans. *Wiener med. Wochenschr.*, 1890, No. 3.

362. Grósz, E. The treatment of detachment of the retina. *Szemészet*, 1890, No. 1.

363. Grünthal. Retinal hemorrhages in intoxication with hydracetine. *Centralbl. f. prakt. Augenhk.*, vol. xiv., p. 73.

(The affection is ascribed to the changes in the walls of the blood-vessels, caused by the destruction of the red blood-corpuscles.)

364. Hess, C. Examination of a case of semilateral disturbance of the color-sense in the left eye. *v. Graefe's Arch. f. Ophth.*, vol. xxxvi., 3, p. 24.

365. Heyne. A case of general cutaneous and sensory anæsthesia. *Deutsches Arch. f. klin. Med.*, vol. xlvii., Nos. 1 and 2, p. 75 ; in the same, a paper by Ziemssen on the same subject.

366. Javal. Scotome scintillant. *Soc. d'opht. de Paris*, July 1, 1890.

367. Lagrange. Contribution à l'étude anatomique et clinique du gliome de la rétine. *Arch. d'opht.*, 1890, vol. x., 5., p. 85.

368. Masselon. Des altérations consécutives aux hémorrhagies de la rétine. *Soc. franç. d'opht.*, May 8, 1890.

369. Naumoff. Some pathologico-anatomical changes in the fundus of the eyes of new-born children. *v. Graefe's Arch. f. Augenhk.*, vol. xxxvi., 3, p. 180.

370. Ottolenghi. The visual field of epileptics (when free from an attack) and of congenital criminals (morally insane). *Centralbl. f. Nervenkrankh. u. Psychiatrie*, 1890, vol. xiii., p. 289.

371. Pröbsting, A. A case of retinitis proliferans. *Zeh. klin. Monatsbl. f. Augenhk.*, 1890, p. 73.

372. Rampoldi. Cecità unilaterale sequita a fissazione del sole in parziale eclisse. Reporto endoftalmoskopico negativo. Inefficazia di alcuni mezzi terapeutici. Guarizione rapida ottenuta colle fumigazioni di fegato di bue. *Ann. di ottalm.*, vol. xix., 3 and 4, p. 263.

373. Schleich, G. A contribution to the development of spontaneous new formation of connective tissue in the retina and vitreous. Retinitis proliferans. *Zeh. klin. Monatsbl. f. Augenhk.*, 1890, p. 63.

374. Schöler. The operative treatment of detachment of the retina by injection of iodine into the eyeball. *Berliner klin. Wochenschr.*, 1890, No. 34.

375. de Schweinitz, G. E. A series of cases presenting minor lesions at the macula lutea. *Journ. Amer. Med. Asso.*, Oct., 1890.

376. Tiffany, Flavel B. Cécité amenée par la quinine ou amaurose quinique. *Rec. d'opht.*, 1890, p. 321.

377. Weissenberg. A contribution to the knowledge of the disturbances of reading based upon a case of dyslexia. *Arch. f. Psych. u. Nervenkrankh.*, vol. xxii., 2, p. 414.

Abadie (356) again recommends injections of iodine (iodo-iodurée) in detachment of the retina. He had a special knife constructed for this purpose, the handle of which forms a Pravaz syringe. Boe is opposed to these injections. In simple detachment of the retina by a subretinal exudation Abadie resorts to puncture ; in complete detachment he does not interfere. According to Guende's report Despagnet injects sublimate (1:2000).
Marckwort.

In opposition to Logetschnikow, who doubts the correctness of the diagnosis of temporary detachment of the retina in albuminuria of pregnant women, with œdema and eclamptic attacks, considering them retinal œdema, Adamück (357) describes a case of binocular, total funnel-shaped undoubted detachment of the retina, which had developed in a woman aged thirty in the eighth month of pregnancy during a severe eclamptic attack—$V = \frac{1}{\infty}$. It rapidly improved after prematurely-induced delivery of the child, which was resorted to ; a few days later, only a slight detachment in the lower part of both eyeballs was found, which a week later had entirely disappeared, with $V = \frac{20}{60}$. As to the development of detachment the author expresses the opinion that it can only take place when the volume of the vitreous has been diminished or ectasia of the sclera has taken place. An injury may break up the vitreous, convert it partly or entirely into free liquid or fibrous tissue (remnants of the supporting tissue), which Adamück experimentally imitated on the vitreous in a rubber bladder, and then only can the retina become detached. When the vitreous is healthy, neither an exudation nor a rent in the retina can cause detachment.
Hirschmann.

The results of Bernhardt's (358) investigations may be summed up by saying that there is not only a general extensive contraction in the diseases in question, but only one color is found with good external limits for white. It resembles the condition of incipient atrophy. This circumstance should make us cautious in utilizing the outlines of the visual field. Sometimes the temporal half or even the whole optic nerve shows a slight pallor ; this need not be considered atrophy, though the visual field may indicate it, for it disappears again.

Mr. N. N., aged forty-three, had two years previously in the right eye a small central scotoma, which soon disappeared. Lately he has had difficulty in reading. O. D. V $= \frac{5}{12}$, O. S. $\frac{5}{6}$. The visual field, when examined in the usual manner, was found of normal extent in both eyes. Leftsided homonymous hemianopsia for colors. When tested with very small white objects ($2\frac{1}{2}$ *mm* diameter, distance 2 *m*), leftsided homonymous hemianopsia was found in both eyes. With the aid of the same test-objects Bjerrum (359) could demonstrate in both eyes a small absolute scotoma to the left of the point of fixation. In the right eye this scotoma gradually extended in the course of the disease beyond the point of fixation into the right half of the visual field. No changes visible with the ophthalmoscope. The author thinks the seat of the disease is at the upper boundary between the right tractus and chiasma. Schiötz.

In Dunn's (360) case a boy of nineteen was struck on the right eye by a snowball. No affection of sight at the time, but six months later it began to fail, and the loss was gradual until it was reduced to $\frac{8}{200}$ in the right, $\frac{18}{70}$ in the left eye. There was also constriction of the visual field and some increase of tension, and the anterior chamber was shallow ; media clear, fundus normal. Pain on exposure to wind and a constant painful winking. Tenderness over supraorbital nerve. He was treated for syphilis and gave up tobacco without benefit. Section of the supraorbital nerve gave instant relief to the painful symptoms, and vision returned gradually to $\frac{18}{15}$ in both eyes. Burnett.

Grósz (362) reviews the methods of treating detachment of the retina, and reports the results of sixty-nine cases treated at the Budapest eye clinic. Injections of tincture of iodine failed.

Hess (364) examined a technicist aged thirty-one, in whose left eye sight and color-perception had diminished. No ophthalmoscopic changes. The power of recognizing red and green had completely disappeared in the nasal half of the retina of the left eye, while that of blue and yellow was only diminished but to the same extent. The power of recognizing black and white was also somewhat impaired.

In HEYNE'S (365) case V in the right eye was $\frac{1}{8}$, in the left $\frac{1}{10}$, 4 D of H and normal fundus; the sense of touch and temperature of the skin and mucous membrane, the sense of pressure and localization, and the perception of pain were paralyzed. The sensibility of the muscles was abolished. The sensibilities were absent, and the functions of all the organs of sense were defective. By closing the ears and bandaging the eyes, thereby removing all excitation, sleep could be produced at once. The patient recovered. The nature of the disease has not yet been discovered.

JAVAL (366) showed the Soc. d'opht. de Paris representations of the scotoma scintillans. They were made by a prominent artist after observations upon himself and show the different stages of scotoma. He adds an accurate description of the scotoma observed upon himself. (Everyone who has had the opportunity of observing the scotoma on himself will agree in declaring both the drawings and the descriptions excellent. —Rev.) MARCKWORT.

LAGRANGE (367) believes that the dangers of glioma are not infrequently overrated. He enucleated the left eye of a boy aged seven for glioma, and the child was still in perfect health two years later. In this case the glioma was an endophytic one, according to Hirschberg's classification, though its origin in the retina could no longer be determined. It occupied the lower outer part of the interior of the eye and had left the anterior part of the eye intact. v. MITTELSTÄDT.

MASSELON (368) discusses the changes following hemorrhages into the retina, especially the fatty degeneration of the parts affected. Such hemorrhages may occur also in progressive myopia, besides in arterio-sclerosis, in conjunction with the fatty degeneration just mentioned. According to Wecker's experience atrophy of the optic nerve often follows extensive retinal hemorrhages. Galezowski opposes Wecker in this respect.

MARCKWORT.

NAUMOFF (369) examined under the microscope the eyes of forty-seven new-born children, who had died during or soon after birth, and found changes similar to those observed in the first stages of choked disc, retinal œdema, infiltration of the retina with a finely granular albuminous substance, and hemorrhages of different size and shape in the retina and choroid. The hemorrhages in the granular layers did not spread in the direction of the radiating fibres, as in adults, but at right angles to them. They are due to rhexis and diapedesis in protracted labor and constricted pelvis, and are the result of the marked choking up of the blood and the increase of the intracranial pressure. It is a curious fact that the hemorrhages are absorbed without leaving larger patches visible with the ophthalmoscope. Afterwards such cases are designated as cases of amblyopia congenita. The so-called congenital atrophy of the optic nerve might sometimes be referred to the change in the optic nerve just described. The central coloboma may also be due to the affections in question.

OTTOLENGHI (370) found in the epileptics and congenital criminals a marked limitation of the visual field. The outer parts in particular are affected, and show a peculiarly ragged outline.

RAMPOLDI (372) reports on a uniocular amaurosis which had developed in a young girl after she had observed the eclipse of the sun on June 17, 1890, for about five minutes through a slightly smoked glass. $V = \frac{1}{\infty}$, pupil immovable, no ophthalmoscopic changes. After numerous remedies had been tried for three weeks without avail, the author subjected the patient for twenty to twenty-five minutes to fumigation with fresh ox-liver, an old empiric remedy in hemeralopia. Recovery rapid. DANTONE.

From the observation of two cases, in which hemorrhage into the retina and vitreous had previously taken place, SCHLEICH (373) thinks that in such cases the development of connective tissue takes place in these hemorrhages from the retinal blood-vessels.

SCHÖLER (374) claims to have obtained eight cures among twenty-eight cases. He includes three cases in which the detachment was still present but sight was much improved.

DE SCHWEINITZ (375) describes some groups of cases in which vision is normal and without known cause some changes are found in the region of the macular lutea, usually slightly above and to the temporal side. They are generally oval in shape, of varying tints, but mostly grayish-white or buff-colored, and occasionally associated with semi-granular pigment. They vary in size from one to four diameters of a retinal vein. The groups are. 1. Asymmetrical lesion situated in the macular region of the eye presenting the greater error of refraction. Ten cases. 2.

Symmetrical mascular changes—symmetrical refractive errors. Four cases. 3. Asymmetrical lesions, situated in the macular region of the eye presenting the smaller error of refraction. Four cases. 4. Symmetrical macular changes, asymmetrical refractive errors ; probable influence of constitutional derangement. Five cases. 5. Slight macular changes, the apparent result of exposure to bright sunlight. One case. BURNETT.

FLAVEL B. TIFFANY (376) saw quinine amaurosis in a man, aged thirty-four, following a dose of 1.95 grm. At first there was complete amaurosis, but improvement gradually took place, so that after sixteen days the patient could read again. No exact statement as to the acuteness of the sight regained is given. The optic nerve and retina were of a whitish color and anæmic. MARCKWORT.

In a fatal case of dyslexia WEISSENBERG (377) found an angiosarcoma in the occipital lobe. He theorizes as to the cause of dyslexia and thinks it may be a purely functional symptom, and that recovery may take place.

XIX.—OPTIC NERVE.

378. DEUTSCHMANN, R. Contributions to ophthalmology Hamburg and Leipzig, L. Voss.

379. HANSEN, C. M. Et tilfcelde af akut retrobulbar neuritis. *Nord. ophth. Tidsskr.*, vol. iii., 2, p. 68. Copenhagen, 1890.

380. JENSEN, E. Om de met central Scotum forlöbende Ciensygdomine. *Nord. ophth. Tidsskr.*, vol. iii., 1, p. 33.

381. MARTIN, J. De l'atrophie du nerf optique et de sa valeur pronostique dans la sclérose de la moëlle épinière. Paris, 1890, Asselin et Houzeau.

382. MOELI, C. Changes in the tractus and optic nerve in diseases of the occipital lobe. *Arch. f. Psychiatrie u. Nervenkrankh.*, vol. xxii., 1, p. 73.

383. SUTPHEN, J. Sarcoma of the optic nerve, notable chiefly for its size and shape. *Trans. Amer. Ophth. Soc.*, 1889.

384. WEYERT. The heredity of coloboma of the optic nerve. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 325.

DEUTSCHMANN (378) reports several clinical histories, and adds some remarks partly on the basis of clinical observation, partly of pathologico-anatomical examination. They concern among others the pathology of the optic nerve and its entrance, homonymous hemianopsia after cranial injury, amaurosis from autointoxication in carcinomatosis, spontaneous detachment of the retina with deepening of the anterior chamber, and some rare forms of tumor of the eye.

Jensen (380) found among 60,125 patients of Hansen's clinic at Copenhagen 259 cases with central scotoma, divided into the following groups: (1) Amblyopia centralis, 189 cases, generally people beyond thirty. Causes, abuse of tobacco and alcohol. There is never an absolute scotoma for white objects when tested as usual with the perimeter. Typical color-scotoma. Vision rarely sinks below fingers at 4 to 5 *m.* Recovery or improvement is the rule; it never ends in atrophy. (2) Stationary scotomatous atrophy of the optic nerve. Twenty cases, men exclusively, usually between the ages of twenty and twenty-five. Hereditary basis, general weakness, or no traceable cause. The disease develops either suddenly or reaches its maximum in a short time without disturbing the general health; it generally manifests itself in both eyes at the same time. There is a central scotoma of the same size and shape as in central amblyopia, but of much greater intensity; fixation is therefore uncertain or eccentric. The periphery is normal. The ophthalmoscope reveals atrophy of the optic nerve, often at an early stage. The prognosis as to restitution is bad, as to blindness absolutely good. (3) Progressive scotomatous atrophy of the optic nerve. Twenty-three cases, men between the ages of forty and fifty, who generally have had syphilis. This disease shows tabic prodromal symptoms, such as immobility of the pupil, myosis, etc. It begins with central amblyopia, color-scotoma with normal periphery. Afterward the color-field contracts and finally also the field for white becomes smaller. It ends in complete atrophy of the optic nerve. It is not an independent disease, but is a special form of development of the tabic atrophy of the optic nerve. (4) Binocular scotomatous optic neuritis. Eleven cases.

According to Martin (381) the development of atrophy of the optic nerve is prognostically of great importance for the further course of tabes. With the beginning of amaurosis the disturbances of motility which have already developed do not disappear, but frequently the pains cease, or at least become less. The tabes remains stationary after the loss of sight.

Marckwort.

Moeli's (382) cases were cases of parencephalitis and hydrocephalus internus in younger persons with atrophy of the optic nerves and the tractus. From this he concludes that destruction of the undeveloped brain, when it affects the white substance of the occipital lobe is combined with atrophy of the optic nerves and tractus. In a third patient, aged forty-four, he found atheromatous degeneration in both occipital lobes, more or less marked changes in the optic-fibre portions of the mid-brain, and only slight degeneration in the optic nerves and tractus. Further investigations must show whether in adults in affections of the occipital lobe changes regularly take place in the mid-brain, whether they spread towards the periphery, etc. No definite conclusions can be drawn from the literature of the subject, as in the affections mentioned changes were sometimes found in the optic nerves, sometimes not.

Sutphen's (383) patient was a girl of ten years, with a movable tumor over the left orbit which extended back into the latter by a pedicle which was found attached to the optic nerve, which was very much swollen. The globe was entirely absent, it having atrophied two years ago as a result of inflammation, probably caused by the tumor. Burnett.

Weyert's (384) were cases of simple coloboma of the optic nerve found in three generations, becoming less marked from the older to the younger children, and from the older to the younger generation. The functions of the eyes were normal.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

385. Coppez. De l'intervention chirurgicale dans les blessures de l'œil avec pénétration des corps étrangers. *Soc. franç. d'opht.*, May 5, 1890.

386. Fano. Un nouveau cas de conservation de l'œil atteint d'une blessure grave. *Journ. d'ocul. et de chir.*, 1890, p. 171.

387. Gast, R. Case of intraocular cysticercus. *Zeh. klin. Monatsbl. f. Augenhk.*, 1890, p. 10. (Extraction through a meridional scleral incision.)

388. Hirschberg, J. On the results of the magnet-operation in ophthalmology. *v. Graefe's Arch. f. Augenhk.*, vol. xxxvi., 3, p. 37.

Coppez (385), as reviewer in Paris, discusses the surgical procedures in cases of foreign bodies in the eye. The following passages are worthy of note: "in a patient, whose eye has been injured by a penetrating foreign body, the attempt must be made to remove the latter as soon as possible with Hirschberg's electro-magnet when it is metallic and there are no adhesions, or with a curette and forceps if necessary, after enlargement of the wound." "When the foreign body has entered through the ciliary region and the eye is threatened with panophthalmitis, enucleation should be done at once." "When the media and lens are clouded, it is best not to interfere, because uncertain manipulations may not only endanger the injured but also the healthy eye." When the foreign body has become encapsuled and causes little irritation, expectant treatment simply is indicated. MARCKWORT.

HIRSCHBERG'S (388) paper is a very readable one, but cannot be briefly reviewed on account of the numerous details gathered from his large experience. He advocates the use of the magnet in all cases in which a chip of steel has been shown to enter or is supposed to be present, with the exception of those in which the eyeballs are free from irritation and the foreign body is firmly fixed in the membranes of the eye. Besides probing with the magnet, best in narcosis, the primary meridional incision is indicated directly after the injury, the secondary during the state of irritation, and the tertiary in case of new inflammation, even though it be after years. The prognosis both for the preservation of sight and the shape of the eyeball mainly depends upon whether the patient comes under treatment soon after the injury, whether infection has taken place, and how large the piece of iron is. His conclusions are drawn from the observation of one hundred cases. By extracting the chip of iron from the vitreous he obtained good, even normal sight in four cases; moderate sight in three; in six others he preserved the shape of the eyeball. He very properly urges that workmen should wear protecting eye-glasses.

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

389. BERGER. Les troubles oculaires dans le tabes et la théorie du tabes. *Rec. d'opht.*, 1890, pp. 195 and 263. (Essentially a French translation of his paper in these ARCHIVES.)

390. BERGMEISTER. The connection between influenza and the visual organ. *Wiener klin. Wochenschr.*, 1890. No. 11. (The observations of atrophy of the optic nerve are new.)

391. BOUCHUT. Du diagnostic des maladies du système nerveux par l'ophthalmoscopie et la cérébroscopie. *Paris Med.*, March 11, 1890, p. 81.

392. CIRINCIONE. Lesioni nervose nella xerosi con emeralopia. *Giorn. di neuropatologia*, 1890, vol. vii., Nos. 4 and 5.

393. EISENLOHR. The diagnosis of affections of the corpora quadrigemina. *Deutsche med. Wochenschr.*, 1890, p. 937.

394. GEILL, M. Badet ablissement te Pelantrengan (Java). Lepraverslag over het jaar, 1889. *Geneeskundig Tydschrift vor Nederlandsch. Indie*, Deel xxx., off. 1 en 2, bl. 47, Batavia, 1890.

395. HAAL. The effect of influenza on the eye. *Correspondenzbl. f. Schweizer Ærzte*, 1890.

396. LANDSBERG. Visual disturbances after influenza. *Centralbl. f. prakt. Augenhk.*, vol. xiv., p. 141. (Neuritic atrophy.)

397. LAQUEUR. A case of binocular embolic iridocyclitis after influenza. *Berliner klin. Wochenschr.*, 1890, p. 816.

398. v. LIMBECK. A case of gunshot injury of the left cerebrum. *Prager med. Wochenschr.*, 1890, p. 558.

399. MENDEL, E. A case of multiple paralysis of the cerebral nerves. *Neurol. Centralbl.*, 1890, p. 494.

400. PERMEWAN, W. Case of localized suppurative meningitis ; with remarks on the cortical representation of the conjugate movements of the eyes and head. *The Lancet*, Aug., 1890, p. 278.

401. SCHRIMER, O. Unilateral total ophthalmoplegia after influenza. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., p. 312.

402. SULZER, E. Visual disturbances due to malarial infection. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxviii., 259.

403. UHTHOFF. Case of dyslexia. *Berliner Gesellsch. f. Psychiatr. u. Nervenkrankh. ; cfr. Centralbl. f. prakt. Augenhk.*, vol. xiv., p. 113.

404. VIGNES. Papillite par influenza. *Soc. d'opht. de Paris*, July 1, 1890.

BOUCHERT (391) reports a case in which an intracranial, right-sided tumor had been diagnosticated during life from choked disc of the right eye. The autopsy revealed a cyst weighing 500 grms. in the right lobe. Under the microscope it proved to be an echniococcus cyst. MARCKWORT.

CIRINCIONE (392) had the opportunity of observing a case of binocular xerosis epithelialis of the conjunctiva, which began with hemeralopia and headache, was followed by various disturbances of motility and sensibility, and ended in complete desiccation of the conjunctiva and purulent destruction of the cornea. From the conjunctival discharge, small excised pieces of conjunctiva, and the nasal discharge, pure cultures of the well-known xerosis-bacillus were prepared on different soils, which, however, when transferred to the conjunctiva and anterior chamber of the rabbit did not prove to be pathogenic. (Cultures from the blood and spleen gave negative results.) As the patient died of an intestinal tumor, the author was able to examine the tissues in question under the microscope. Nothing abnormal was found in the eyeballs, optic nerves, or brain, but both Gasserian and ciliary ganglia showed marked changes as to their size and the arrangement of the ganglion cells and nerve fibres. The author therefore concludes that the symptoms, repeatedly observed during xerosis, of partial and general dryness and insensibility of the skin of the face, of the cornea and conjunctiva, the ozæna, the hemeralopia, and the diminished tension of the eyeball cannot be attributed to the non-transferable bacillus of xerosis, but primarily to changes in the nerve-centres of the fifth nerve. DANTONE.

EISENLOHR (393) reports on a case of injury of the corpora quadrigemina of the right side by a revolver bullet, which, having healed in, caused a series of successively developing symptoms of central lesions. The autopsy confirmed the diagnosis. The right pupil was wider than the left—both reacted slowly. Sight gradually diminished, without ophthalmoscopic symptoms. Visual field and color-perception normal. Later paralysis of the associated muscles and unsteady gait (cerebellar ataxia) developed· Finally pressure on the brain manifested itself, with stupor, choked disc, and increase of the paralysis of the ocular muscles.

A symptom which frequently occurs in leprosy is the inability to close the lids, so that the cornea frequently suffers. Another cause of the ocular inflammations GEILL (394) finds in the formation of nodules in the iris. In a patient a neoplasm, which covered the pupil and reduced V almost to 0, developed in the iris of the right eye. An iridectomy improved it to $\frac{6}{18}$. After two months many new nodules had developed which closed the coloboma. The neoplasms do not occur at the periphery, but in the central part of the iris. They are of a brownish-red color and are bounded by normal tissue. No development of blood-vessels was observed. Atrophy of the optic nerve is rarely found as a fourth cause. Among twenty-nine patients sight was found normal in only thirteen.

LAQUEUR (397) reports the case of a woman aged forty-three who had suffered with influenza, and who seven days after its beginning became blind in half an hour in both eyes, sight being reduced to perception of light. Acute iridocyclitis with plastic exudation was found. Bacteriological examination of the aqueous gave a negative result. Recovery under atropine and hydrargyrum.

VON LIMBECK'S (398) patient was noteworthy on account of her disturbances of speech. She can only pronounce the syllable ma. Her mental power is not impaired. To decide whether the homonymous hemiopia in question was due to a drag by the injury of the occipital lobe the test applied by Sattler to the so-called hemiopic pupillary reaction was tried ; it succeeded, and so an affection of the occipital lobe was assumed.

In MENDEL'S (399) patient, aged twenty-seven and free from syphilitic taint, paralysis of the oculomotor and facial nerves developed in the course of two weeks. Injury, syphilis, tuberculosis, and diphtheria are the usual causes. These, however, were absent, and so a rheumatic paralysis was diagnosticated on account of the rapid recovery.

PERMEWAN'S (400) case was one of traumatic meningitis, accompanied with convulsions. At the beginning of each fit the eyes and head turned slightly to the left for a short time, and then followed a marked deviation to the right, which lasted to the end of the fit. On post-mortem examination there was found suppurative meningitis over the whole of the left frontal lobe, the area of greatest inflammation corresponding with the lowest part of the middle and lowest frontal convolution, Ferrier's centre for conjugate deviation. The two different movements were probably due to bilateral representation in the cortex.

WERNER.

In SCHIRMER'S (401) patient, a woman, aged fifty-four, there was complete paralysis of the oculomotor, trochlearis, and abducent nerves, and incomplete paralysis of the fifth and facial nerves. The cause was sought in a hemorrhage into the middle cerebral fossa.

In malaria cachexia, SULZER (402) observed the following ocular affections : (1) chronic optic neuritis, (2) diffuse infiltration of the vitreus, (3) numerous small peripheral hemorrhages into the retina, (4) sudden incurable blindness, probably due to central hemorrhages or emboli. During the acute attacks of fever periodic amblyopias of varying degree are observed, sometimes increasing to blindness, marked hyperæmia of the papilla and retina, with loss of the retinal lustre and disposition to affection of the macula lutea from reflected sunlight, and large hemorrhages near the papilla and macula.

In Uhthoff's (403) patient, a woman, aged seventy-two, there was rightsided hemianopsia, with the symptoms of dyslexia, disturbances due to a blow with the fist upon the temple. The patient wrote fluently, but soon after could not read what she had written. Memory had also suffered.

Vignes (404) describes a case of uniocular choked disc due to influenza. The ophthalmic trouble developed on the eighth day of the disease, entirely depriving the eye for some time of perception of light. V however gradually rose again to $\frac{4}{10}$. Galezowski saw four similar cases. Valude observed phlegmon of the orbit during an attack of influenza. Gillet de Grandmont found during the disease a condition resembling coma, with ophthalmoplegia externa. Parinaud thinks that the choked disc in the case of Vignes might have been due to rheumatism, instead of influenza. Despagnet defends the opinion of Vignes.

Marckwort.

MISCELLANEOUS NOTES.

A.—BOOK REVIEWS.

Text-Book of Ophthalmoscopy. By EDWARD G. LORING, M.D., Edited by FRANCIS B. LORING, M.D. Part II., Diseases of the Retina, Optic Nerve, and Choroid : Their Varieties and Complications. Pp. vi + 253. New York, D. Appleton & Co., 1891. Price, $5.00.

It is a melancholy duty that compels us to record this volume as only a fragment—never, alas ! to be completed—of a *magnum opus* of one of America's most accomplished and energetic ophthalmologists—a work which, if it had been carried forward to its termination in the manner which was followed from the beginning up to the point where death stilled the hand of the writer, would have found a place in the front rank of ophthalmological literature of the time.

The present volume was to have contained the use of the ophthalmoscope as applied to the study of the diseases of the interior of the eye, though of its definite scope and limits we have no intimation. If, however, we may judge from what is here presented in a state which we are allowed to consider as complete, the whole subject would have been treated in a very exhaustive manner. By exhaustive we do not mean a mass of heterogeneous details. It was not Loring's method to consider his subjects in that way. He seems to have had a fear—which, within limits, is certainly wholesome and to be commended—of wearying his reader and drawing attention away from the essential features by an introduction of those details which have no bearing on the main issue. His artistic qualities are those rather of the bold etcher than of the miniature painter ; and because of these qualities we are led to doubt whether his work will ever be so acceptable to the beginner as to the studious practitioner. To the latter, however, it will ever be a delight and ofttimes referred to as a help in time of need.

Loring studied ophthalmoscopy with the enthusiasm of an artist, and that enthusiasm he has carried into his book, and it will be shared by the reader who is at all in sympathy. Of few treatises can this be said.

To come to a more detailed examination of the volume, we find that its broad divisions are into: 1st, Diseases of the Retina; 2d, those of the Optic Nerve; 3d, those of the Choroid. Of these the first, we are led by the editor to infer, was complete, and represents the author's views as he intended them to go to the public. It occupies just two thirds of the book, and shows the author at his best. The study of the vascular system of the retina and the changes it undergoes under various morbific conditions is masterful. We cannot recall anything of the kind that equals it in clearness of conception or crispness and succinctness of statement, and in these particulars it is a model for scientific writing of its kind. We regret to find, however, no notice, either in this or in the previous volume of that striking ophthalmoscopic appearance known as "shot-silk retina." We would have been glad to know how so experienced an observer as Dr. Loring regarded this condition, whether as physiological or pathological, there being reasons, at least in the opinion of the reviewer, for considering it either or both.

Of particular interest is his study of the conditions of embolism and thrombosis of the retinal vessels. His own views, as are well-known, incline to the greater frequency of thrombosis as compared with true embolism.

The chapters on the optic nerve are not so complete or satisfactory, and it was evidently intended to give them greater elaboration before publication. The chapter on the choroid is hardly more than some memorandum notes which the editor found and printed without revision. But even these notes are valuable and suggestive, for Loring was a clear as well as an original thinker, and it is always apparent that he saw things at their relative values and not as isolated and independent facts. In this connection we will quote the following sentence closing the chapter on "Irritation of the Retina":

"The fact that troubles in the nasal passages give rise to functional disabilities of the eyes has lately received a new impetus from the observation and success of various practitioners who, with good sense, have turned their attention from the prescribing of glasses of almost infinitesimal degrees of curvature to the medication of the real seat of the disease."

The mechanical get-up of the volume is, like that of its fellow, elegant and a credit to the publishers. The drawings, most of which are from the hand of the author, are good; but, on the whole, we think those in black and white better than those in color.

BURNETT.

Die Entstehung der Entzünding, etc. (The Origin of Inflammation and the Action of Phlogogenic Agencies, studied by Experiments chiefly on the Eye.) By THEODOR LEBER, Professor of Ophthalmology at Heidelberg. With eight lithographic plates. Leipzig. W. Engelmann. 1891.

In this superbly printed volume of 535 in quarto pages the author has presented not only ophthalmology, but general medicine with a work of fundamental importance, the fruit of eleven years of uninterrupted research. It would be out of place, should we attempt to give here a full abstract of its superabundant contents. An enumeration, with short notes, of the different subjects which the author has so thoroughly, exhaustively, and originally investigated, and of which from time to time he had previously published the results, will convince all of our readers that a conscientious study of the book will place them on the most advanced standpoint of modern medical science, assist them in the understanding of numerous pathological phenomena, and give them a multitude of useful hints as to the rational management of a host of obscure, intricate, and so often deleterious inflammatory processes.

The *first part* treats of the inflammation produced by *moulds.* A unique case of ulcerous keratitis in man—a farmer's girl whose cornea had been injured by an ear of oat—showed (1879) the particles of necrosed cornea pervaded by a dense mycelium of aspergillus fumigatus, as was ascertained by pure cultures. This material Leber used for an extensive series of experiments on rabbits, studying and depicting (on four plates) the various symptoms and phases of corneal ulceration. Then he injected emulsions of aspergillus spores into the anterior and vitreous chambers. The consequences were the most varied pictures of corneal necrosis, fibrinous and aseptic puriform exudation, hemmed in by a girdle of leucocytic infiltration.

Experiments with other mould species were only partially successful, or altogether negative, for instance, with penicillium glaucum the temperature maximum of which being below that of the living body.

The *second part* contains the practically far more important inflammations produced by fission-fungi. Inoculations of septic bacteria, of staphylococcus aureus and albus, and leptothrix buccalis.

The *third part* describes the author's extensive investigations on the phlogogenic properties of extracts of certain fungi and their active principles. He finds that they produce suppuration and necrosis under symptoms identical with those of mycotic inflammation. Sterilized (by boiling) suspensions of staphylococcus produced fibrino-purulent exudations easily, but the author had a good deal of trouble in obtaining an active extract that was free from bacteria. By dialysis and filtration through a large quantity of paper under aspiration he at last succeeded in obtaining, free from cocci, a watery solution possessing the same phlogogenic properties as the bacterial emulsions. The condensed (dry) extract was introduced into the anterior chamber of rabbits, and produced the same inflammation, only in somewhat diminished intensity, as the introduction of cocci. The pus formed by the extract was macroscopically and microscopically like the pus produced by cocci, but cocci were never detected in it.

Treatment with alcohol and ether led, after a rest of eight months, to the formation of a crystalline substance with marked phlogogenic properties, on account of which Leber called it *phlogosin.* The chemical and physical properties of phlogosin, as well as numerous experiments made with it, form the conclusions of this very interesting chapter.

The *fourth part* treats of the phlogogenic action of *chemical* substances and foreign bodies (gold, silver, glass, iron, steel, copper, lead), in various parts of the eye. Furthermore, the action of metallic mercury and its salts, and of arsenious acid, moreover a number of organic substances—gamboge, croton oil, turpentine oil, cantharides, jequirity, indigo, uric acid, and indifferent substances as starch, etc., have been experimented with very extensively.

The *fifth part* contains experiments and remarks on traumatisms and subtile insoluble powders as causes of inflammation. Section XXXIV., "On the accumulation of leucocytes at the place of the inflammatory irritation, as the result of chemotactic influences of the phlogogenic substances," shows the author as a staunch adherent to Conhneim's theory of inflammation. This part contains further : The causes of emigration of leucocytes from blood-vessels ; the origin of pus in the anterior chamber in purulent keratitis ; tissue proliferation by chemical irritation ; purulent maceration and destruction of tissue.

The book concludes with remarks on the cause and object of inflammation, and the protective provisions of the organism to ward off assaults. Considering the enormous material of original experimental investigation which practically constitutes a thorough revision of one of the most important subjects of general pathology, inflammation, we cannot too urgently recommend the study of Leber's eminently scientific work. The innumerable contributions to the knowledge of the same subject, scattered through books and periodicals in all civilized languages, are extensively quoted and sifted, by which the volume before us obtains a value only inferior to its own intrinsic merit. H. K.

Oculist's Index Rerum, arranged and classified for the Recording of Ocular Diseases, Errors of Refraction, and Surgical Operations. By S. C. AYRES, M.D., Cincinnati. J. H. CHAMBERS & Co., St. Louis. Price, $3.

This well-gotten up blank-book for indexing ophthalmic cases will be of the greatest help for those who in the preparation of papers are in the habit of supporting their views by statistics from their own practice. A special part is devoted to the records of cataract operations. Space is left at the bottom of each page for Reading References and other cases. The book is useful, not only in preparing papers for societies or publication, but also for collecting the material of instruction furnished by our own experience, showing how many cases of the different diseases have come under our care. It is evident that the "Index" is a most desirable supplement to every oculist's daily case-book.

H. K.

Les Annales d'Oculistique, formerly edited and owned by the late Dr. Warlomont of Brussels, have, by public sale, passed into the hands of Drs. Valude and Sulzer, who will continue their publication in Paris. The editors promise that the *Annales* will appear regularly in monthly numbers of 64 or 80 pages, containing original papers, reports of societies, and early abstracts of the current ophthalmological literature. Address 134 Boulevard, St. Germain.

Drs. Nuel and Van Duyse, for many years connected with the *Annales*, announce that they will continue the publication of this journal in Belgium under another title to satisfy the law. The chief editor will be Dr. J. F. Nuel, professor of ophthalmology in the University of Liège. His address is 28 Rue Louvrex, Liège, Belgium.

B.—OBITUARY.

Dr. George Salmon Norton, of New York, died January 31, 1891. He was born in New Marlborough, Mass., in the year 1851. He graduated at the N. Y. Homœopathic Medical College in 1872. He was appointed surgeon to the (homœopathic) N. Y. Ophthalmic Hospital in 1875, and professor of ophthalmology in the N. Y. Homœopathic Medical College in 1886, on the death of Dr. Liebold. "When the (Homœopathic) *Journal of Ophthalmology, Otology and Laryngology* was established (two years ago) it was most natural that he should be called to the chief editorship, and to his indefatigable energy and industry its unparalleled success has been in a great measure due." He was a well-informed, judicious, and painstaking oculist. "As a diagnostician he was nearly faultless. At the N. Y. Ophthalmic Hospital his deliberate opinion on any doubtful case went unquestioned ; he was regarded as a court of last resort. As a prescriber he was careful, able, accurate, and homœopathic." He has contributed to the literature of ophthalmology a number of valuable papers, some also in these ARCHIVES. The journal which he founded is continued under the leadership of CHAS. DEADY for ophthalmology and otology and MALCOLM LEAL for laryngology. H. K.

Dr. Wm. F. Smith, formerly of San Francisco, Cal., and for the past six or seven years a resident of Chicago, died April 7th last of septicæmia. Born in Hancock Co., Ohio, in 1845, he first studied medicine with Dr. E. Williams of Cincinnati, and there acquired that love for ophthalmic surgery which he practised so extensively in after life. Graduating from Miami Medical College as valedictorian in 1868 he shortly afterward went to Europe where he became a pupil of Becker, Helmholtz, and de Wecker, having been for some time *chef de clinique* of the latter. In 1870 he returned to the United States and began special practice in San Francisco. In a few years he had the largest *clientèle* on the

Pacific coast, was one of the founders of the *Western Lancet*, and became attached to the chairs of opthalmology in Cooper Medical College and in Toland Medical College—the latter now a department of the University of California.

In 1884 he decided to leave San Francisco, and after a short sojourn in the City of Mexico came to Chicago. In 1884–5, under the direction of the Chicago School Board he examined the eyes of 2,500 school children—the first time such an examination had been made in that city. During his life in Chicago he was appointed ophthalmic surgeon to Cook Co. Hospital, and to the Alexian Bros. Hospital.

Although his time was largely occupied with an extensive private practice he paid a great deal of attention to the current and other literature of ophthalmology and otology, and his library was very extensive. His knowledge of Spanish, Italian, French, and German enabled him to keep acquainted with the best works in these languages. His love of mathematics is evidenced by a well-thumbed copy of Helmholtz's "Physiologische Optik," which he declared contained all the so-called "new" formulæ which writers were from time to time putting forward.

Dr. Smith made a number of contributions to the ARCHIVES OF OPHTHALMOLOGY, including a description of his own operation for entropion, and of a fairly successful case of corneal transplantation.

At the time of his death he was engaged in working out a new theory of accommodation, which cost him much thought and labor. Had he lived he would certainly have elaborated it in the direction of overcoming some of those difficulties which the old theory certainly presents. C. A. W.

C.—NEWS.

The *German Ophthalmological Society* will meet at Heidelberg, September 14th, 15th, and 16th.

The Second Triennial Session of the Congress of American Physicians and Surgeons will be held at Washington, D. C., from September 22d to September 25th. The meeting of the Ophthalmological Society will be at the Arlington House, September 23d and 24th.

Dr. Dufour has been appointed Professor of Ophthalmology in the Medical Faculty of Lausanne, Switzerland.

Tab. III.

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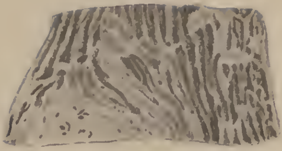

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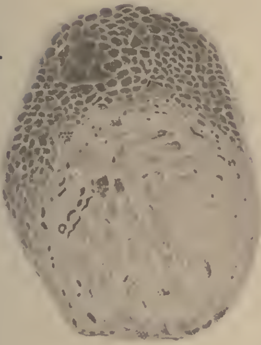

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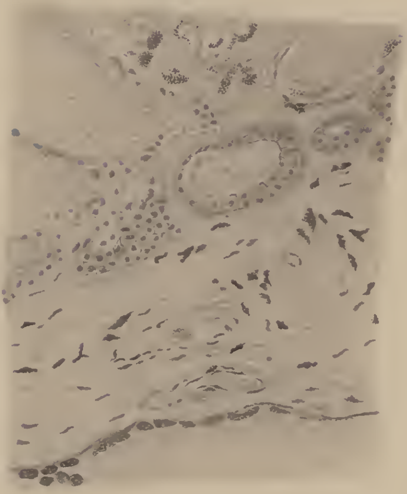

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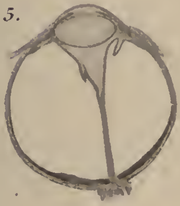

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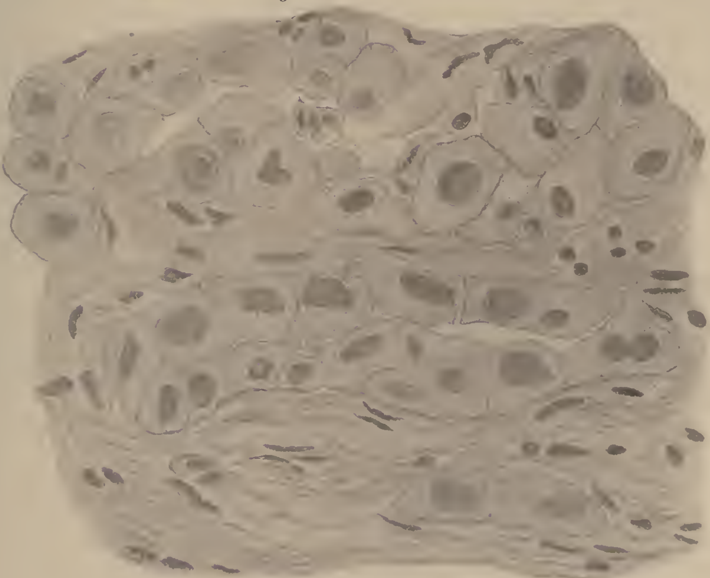

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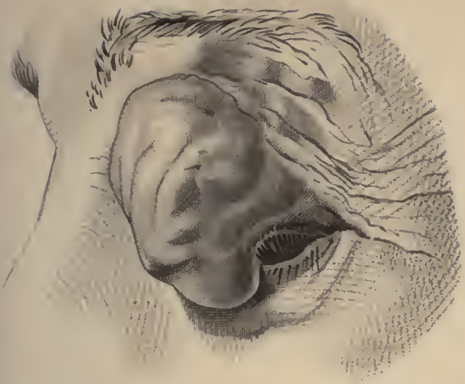

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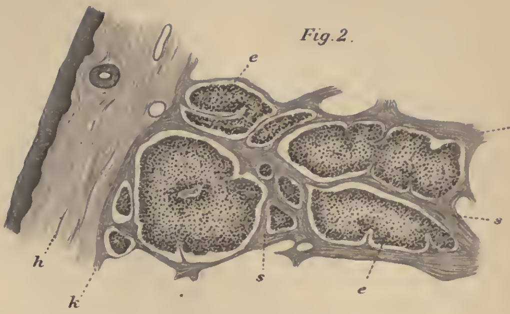

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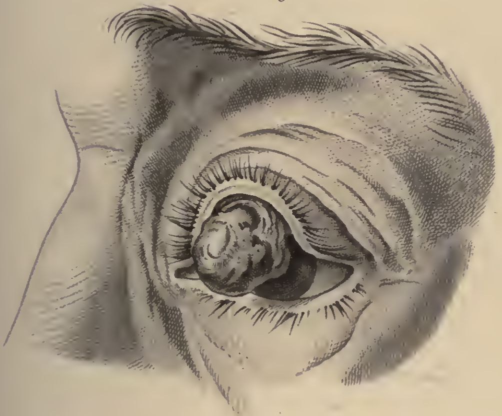

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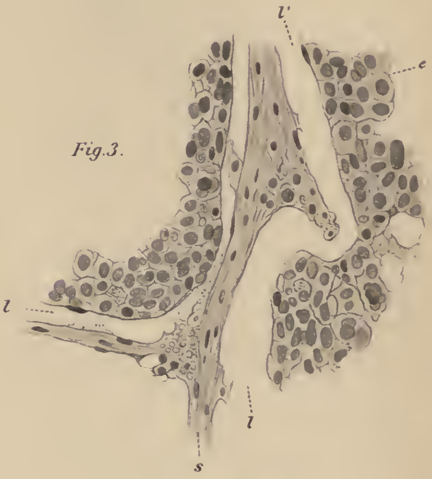

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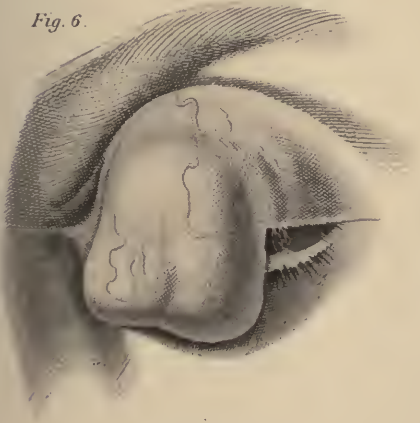

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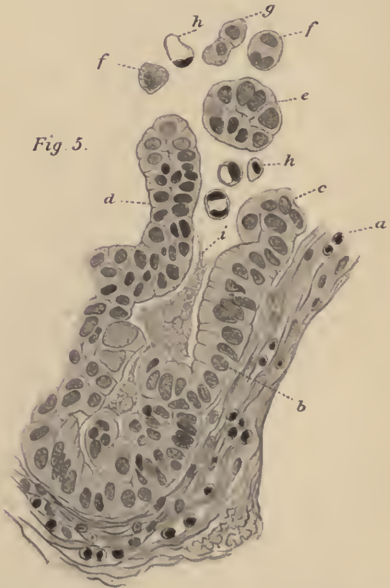

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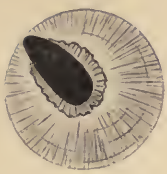

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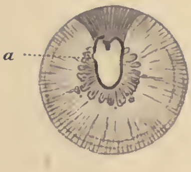

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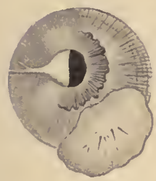

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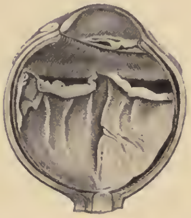

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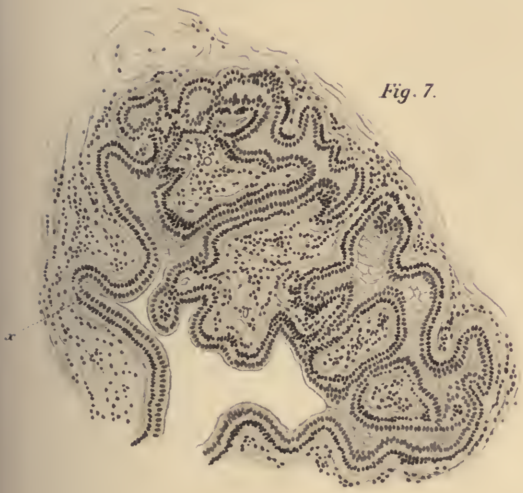

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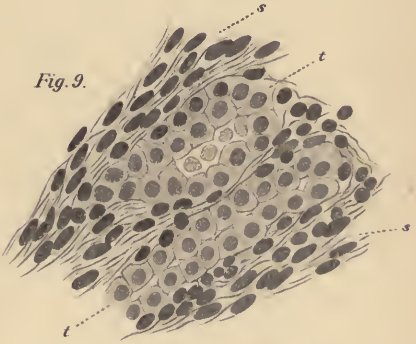

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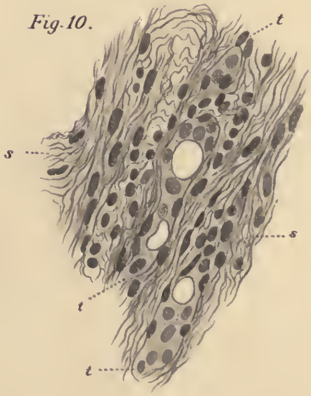

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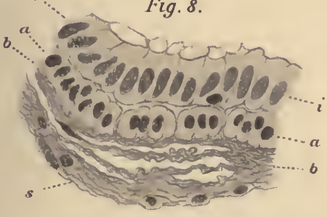

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ARCHIVES OF OPHTHALMOLOGY

NOTICE TO OUR READERS.

The ARCHIVES OF OPHTHALMOLOGY contain *original papers* on all branches of ophthalmic surgery, and *original reports* on the progress of ophthalmology. The original papers occupy from two thirds to three fourths of the space, and their scope embraces all subjects of scientific and practical interest connected with this department of medicine.

The object of the ARCHIVES is to guard, solidify, extend, and diffuse the stock of acquired ophthalmic knowledge by recording the constant addition of new observations, investigations, methods and means of examination, clinical experience, instruments and remedies, and further by taking part in the discussion of the questions of the day with a spirit alike conservative and progressive.

Particular attention is paid to the preparation of the Report on the Progress of Ophthalmology. The report is intended to furnish a *complete*, *systematic*, *and early* review of the current ophthalmological literature of the world. It has been necessary to divide the work of its preparation among a number of collaborators according to the following arrangement :

Dr. ST. BERNHEIMER, of Heidelberg.

1. *General Ophthalmological Literature :*
   - *a.*—Text-books (not abstracts, but notes pointing out what is new and characteristic).
   - *b.*—Monographs and papers on historical, statistical, and general subjects ; reports on societies and hospitals.
   - *c.*—Periodicals on our specialty (short notices of their character, time and place of publication, the titles of the original papers, with the names of their authors).

2. *General Pathology, Diagnosis, and Therapeutics*, including new instruments, apparatus, and remedies.
3. *Normal Anatomy and Physiology*, including color-blindness.

Dr. C. Horstmann, Prof. at the University of Berlin (Potsdamer Strasse 4):

1. *Anomalies of Refraction and Accommodation.*
2. *Eyelids.*
3. *Lachrymal Apparatus.*
4. *Muscles and Nerves.*
5. *Orbit and Adjacent Cavities.*
6. *Conjunctiva, Cornea, Sclerotic.*
7. *Injuries and Foreign Bodies of these parts.*

Dr. P. Silex, Lecturer at the University of Berlin.

1. *Uveal Tract*, including glaucoma.
2. *Refracting Media*, including the lens.
3. *Retina and Optic Nerve.*
4. *Injuries and Foreign Bodies of these parts.*
5. *Ocular affections in connection with general diseases.*

In order to obtain complete and recent reviews on the non-German ophthalmological literature, special reporters have been selected for different countries, who will send monthly abstracts of all local ophthalmological publications to Dr. C. Horstmann, of Berlin, the editor of the Report. Dr. Horstmann will distribute these abstracts among the German reporters according to the above plan, in order to preserve the systematic arrangement which we think of paramount importance.

The special reporters are:

For America: Dr. Swan M. Burnett, 1770 Massachusetts Ave., Washington, D.C.

For Great Britain and Ireland: Dr. L. Werner, 27 Upper Merrion Street, Dublin.

For France and Belgium: Drs. E. Marckwort, Antwerp, and P. von Mittelstädt, Metz.

For Germany: Drs. BERNHEIMER, Heidelberg, HORSTMANN, SILEX, and DEUS, in Berlin.

For Italy: Dr. DANTONE, Rome.

For Russia: Prof. HIRSCHMANN, Charkow.

For Scandinavia: Drs. OLE BULL and SCHIÖTZ, Christiania.

For Holland: Dr. C. A. H. WESTHOFF, of Amsterdam.

In the interest of our readers, who are scattered over the whole world—and whom, like ourselves, we desire to keep informed of all advances in ophthalmology,—furthermore in their own interest, authors of ophthalmological works are requested to send reprints, duplicates of journals, or copies for reviewing, to one of the above-named reporters, or to the editors or publishers of the Archives, according to their convenience.

The abstracts of American ophthalmological literature formerly appeared under a special head, but are now embodied in the systematic Report. Text-books may be specially noticed.

The number of good papers offered us for publication has increased to such an extent that it is no longer possible to translate all of them unabridged. Whenever a German paper is condensed in the English version, or *vice versa*, it will be so stated. Should any one of our readers wish to recur to the original, the editors will always take pleasure in sending it for reference.

Under the heading of "Miscellaneous Notes," there will be published all kinds of professional news which specially concern the oculist, *e. g.*, appointments, honors, resignations, vacancies, new ophthalmic hospitals, opportunities for instruction, prize questions and essays, announcement of Society meetings, etc. Brief notices of this kind will be thankfully received by the editors.

Original papers of value from any source will, as heretofore, be welcome to the ARCHIVES, and are solicited.

EDITORS:

H. KNAPP, M.D.,
25 WEST 24TH STREET, NEW YORK.

C. SCHWEIGGER, M.D.
ROONSTR. 6, BERLIN

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NOTICE TO CONTRIBUTORS.

The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with their contributions.

1. As original communications the ARCHIVES can accept only such papers as have never been printed nor are intended to be printed in other journals. If a preliminary communication on the subject of a paper has been published, the author is requested to state this in the letter accompanying his manuscript. It is understood that contributors to these ARCHIVES and editors of other periodicals will make no abstracts of the original papers published in this journal without giving it due credit for the same.

2. Authors will receive gratuitously twenty-five reprints of their articles. If a greater number is desired,—notice of which should be given at the head of the manuscript,—only the additional cost of presswork and paper will be charged to the author.

3. In preparing manuscript for the compositor it is requested that the following rules be adhered to:

a. Write on one side of the paper only.

b. Write without breaks, *i. e.* do not begin a new sentence on a new line. When you want to begin a new line or paragraph at a given word, place before it in your MS. the sign ¶.

c. Draw a line along the margin of such paragraphs as should be printed in smaller type—for instance, all that is clinical history in reports of cases, etc.

d. Words to be printed in *italics*, should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times.

4. Authors may receive proofs for revision if they will kindly return them without delay. We beg however to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them, to make, if possible, no alterations at all in their MSS., or, at least, to limit these to what is of essential importance.

VOL. XX. No. 4.

ARCHIVES OF OPHTHALMOLOGY.

GLAUCOMA AND AFFECTIONS OF THE OPTIC NERVE.

BY PROF. C. SCHWEIGGER, BERLIN.

Translated by Dr. CASEY A. WOOD, of Chicago.

(*With Twenty-one Wood-cuts.*)

IN his extensive treatise on glaucoma[1] von Graefe laid stress upon the differences between that disease and certain other forms of visual disturbance, which he designated "amaurosis, accompanied by excavation of the optic papilla." I shall quote some of his statements bearing upon this subject, as I consider them as still entirely true: "In some cases which had been diagnosed as glaucoma after an ophthalmoscopic examination only there were no external indications of the glaucomatous condition to be found. The alteration in the nerve itself constituted the only evidence of the disease." "In the absence of other symptoms of increased tension we are not justified in regarding such affections of the optic nerve as a direct result of intraocular pressure. It seems to me that it is absolutely necessary, if we are to understand these cases, that they should be removed from the category of glaucomatous diseases, and studied under the above-mentioned or some other title." "Although from my own experience I have learned that there is much uncertainty in studying these matters, yet I cannot too strongly insist that in the present state of our knowledge the *optic-nerve affection alone is not sufficient to warrant a diagnosis of glaucoma; in many instances it has a pathogenesis entirely distinct from the latter disease.*"

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[1] *v. Graefe's Archiv*, 1857, Bd. iii., 2.

Unfortunately, however, the study of these affections has not proceeded upon the lines laid down by von Graefe, but under a new name has been carried on in quite another direction with anything but fruitful results. In 1861 (with Donders' approval) von Haffmann's[1] article appeared. In it typical *glaucoma simplex* is described, and the inflammatory manifestations of glaucoma are considered as a complication of the disease. Nothing is said about amaurosis associated with excavation of the disk.[2] "Experience has taught us that in every instance where the characteristic form of the excavation is present, *and it stretches to the margin of the papilla* even when no increased tension has been discovered, the latter must be considered, if not as the morbid condition itself, at least as the chief sign of it. It must be remembered that many symptoms of increased tension may be absent. The skilled finger will, however, immediately and unerringly discover the presence of increased tension. It may be present in only a slight degree and have very slowly developed." "The difference between von Graefe's observations and our own is that we believe increase of tension, more or less marked, always accompanies the typical excavation just referred to. We might second von Graefe's contention had we been able to find a case where such a papillary excavation was present without increased tension."[3]

It is not too much to say that von Graefe may be trusted to have examined with "skilled finger" his cases of amaurosis with optic-nerve excavation. [4] In the same way it is now conceded by every one that cases of excavation reaching to the margin of the disk are not uncommon where the skilled touch has been unable to detect any trace of increased tension. The candid, unprejudiced observer must admit that increase of intraocular pressure, however important its pathological significance, must be carefully considered in the light of other diagnostic evidence. Augmented tension and glaucoma are by no means identical conditions. I have, for example, seen the former occur temporarily in severe attacks of iritis, and yet, although observed for years afterwards, the eye presented no sign whatever of glaucoma. In many such cases I have been unable to satisfy myself that the increase of tension had not been produced by spasmodic contraction of the ocular muscles when the eyeball was pressed upon. Moreover, I have observed unilateral plus tension as a temporary manifestation in eyes unaffected by either inflammation or glaucoma. On the other hand, increase of tension may, *just at the time of examination*, be doubtful or altogether absent in cases where glaucoma was certainly present. It often happens that a satisfactory estimation of the ocular tension is rendered unsatisfactory owing to spasm of the orbicularis and the other external muscles, having been produced by the ordinary tactile examination. There is no such thing as a standard of hardness by which the tension of all eyes can be determined. The sense of resistance conveyed to the finger depends not only upon the intraocular pressure, but upon the rigidity of the sclera, which varies not only in individuals but even in different parts of the same eye. This is especially true of myopes, where in the posterior half of the globe there is more or less stretching and thinning of the sclerotic. There are eyes that are physiologically hard and eyes that are physiologically soft. The most reliable method of all is to compare the hardness of the two eyes, but even then it is often difficult in abnormal cases to decide whether there is increase of tension or not. Indeed, the diagnostic value of this "objective" examination is often affected by subjective factors. Whenever an observer decides that there *ought to be* increased tension, his "practised finger" will surely feel it. And then follows the determination of the particular degree of tension with a T + 1, + 2 or + 3! Does any one imagine that two independent examiners would be sure to report T + 2 in the same case? The results are very little better with the tonometer and with manometric measurements.[1] In my opinion the best plan to determine the ocular resistance is that of Coccius—to press directly upon the sclera. Patients who make this method of examination impossible by spasmodically squeezing their lids together will do the same thing if you attempt to touch their eyelids.

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[1] *L. c.*, Bd. viii., 2, S. 124.
[2] *v. Graefe's Archiv*, Bd. viii., 2, S. 147.
[3] *L. c.*, Bd. viii., 2, S. 150.
4 cf Fuchs p 372 [illegible] 8

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[1] See, for example, *v. Graefe's Archiv*, Bd. xxix., 2, S. 11, and Bd. xxxi., 1, S. 105. In the former place atropine is found by manometric measurement to *increase*, in the latter to *diminish*, the intraocular pressure!

The weakest evidence of all is the "typical" excavation. Every excavation which reached the papillary margin was without more ado set down as glaucomatous, and every case which von Graefe was careful to specially designate as amaurosis with optic-nerve excavation, was labelled glaucoma simplex. Of course in this category of von Graefe's were included not only cases of true glaucoma, but also others whose excavation-sign had quite another significance. But that was just the task which he had set himself—how to distinguish, by means of further observation, the one class from the other. However, the "typical" excavation theory carried the day; everybody was converted to the new gospel, and it is not an over-statement to say that they then became a prey to tactile hallucinations, inasmuch as no one now denies that deep papillary excavations, as well as those that extend to the border of the disk, are not infrequently present without the least sign of increased tension.

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If we inquire into the conditions under which such excavations occur without glaucoma, we must begin with a study of the physiological excavation. Our knowledge of the physiological leads us to an appreciation of the pathological. That the optic-nerve fibres may reach the plane of the retina they must necessarily make a rectangular bend; consequently there is always a small pit in the centre of the papilla, which is usually of a lighter color than the rest of the disk, and often presents the form of a funnel. Its width and depth vary greatly; it may involve most of the papillary surface, and so be wide and shallow, or it may be quite small and deep.

To draw the line somewhere, I might define the term physiological excavation to be one which includes more than one third of the disk surface, and whose floor is formed by the lamina cribrosa. If one discovers such an excavation in one nerve, he may confidently expect to find a similar pit in the other. There may be slight variations in their breadth and depth, especially in those large excavations that involve more than half the surface of the papilla, and approach its margin. In several of these instances of large physiological excavation I have been able to trace a hereditary transmission. I do not believe that they are associated with any particular refractive condition, hypermatropic or other. Frequently the bottom of the excavation (the lamina cribrosa) lies considerably behind the plane of the choroid.

When a physiological cavity includes more than one half the disk surface and the lamina cribrosa lies deeper than usual, the fibres that issue from the middle of the nerve trunk hug the walls of the excavation, and do not make their rectangular *bend* until they reach the papillary margin. Consequently the nerve fibres that supply the medial half of the retina constitute the medial border of the cavity, while those fibres that go to the temporal part of the retinal periphery form its upper and lower margins because the temporal border is reserved for the macular fibres. This is also true of the blood-vessels. The temporal border of the physiological excavation in consequence of this presents, in most cases, a lighter coloration and (as may be seen by observing the small vessels that run towards the macular region) a less steep declivity. The medial border of the physiological cup, on account of its rich vascular supply, is of a deeper reddish-gray color, and in consequence of the above-described disposition of its nerve fibres usually assumes a semilunar shape. In the case of a large excavation its margin is almost identical with the edge of the disk, since the layer of nerve fibres at the retinal border is only 0.2 *mm* thick, about the same thickness as the layer which lies between the excavation margin and the border of the sclerotic. If we consider the diameter of the optic nerve to be 1.5 *mm*, then the thickness of the nervous layer at the papillary border is about one eighth of the whole. In the ophthalmoscopic picture it looks as if the papillary margin and border of the excavation were one and the same. This is not always true of its entire circumference, but it is very often the case with the upper border of the excavation. The larger retinal vessels project at the border of the physiological pit, and then bend abruptly like a hook. While they are closely applied to the wall of the pit, they are visible either in foreshortened perspective, or are not seen at all. Inasmuch as in an ophthalmoscopic examination the observer is confined to the view obtained in a straight line only, it can happen that the overhanging medial border of the excavation may hide, or almost hide, the point at which the retinal vessels are given off.

All these conditions obtain in glaucomatous excavations, and for the same reason. When, as in elderly persons, the retina loses its lustre and transparency, this resemblance to pressure-cupping becomes still more marked. Indeed cases have come under my observation where solely on account of such excavations eminent oculists have subjected their patients to iridectomy, and have even produced in them traumatic cataract, while certain others, whose physiological excavations were stated to call for urgent operation, only saved themselves by immediate flight. These latter, remaining under my observation for years, continued to have normal visual acuteness, and to retain an unrestricted visual field.

When there is joined to a pre-existing physiological excavation an optic nerve atrophy, the difficulties of diagnosis are greatly increased. Müller[1] has pointed out that in the latter case connective-tissue remains are visible on the lamina cribrosa posterior to the retinal plane—a condition which he describes as atrophic excavation. I have pictured such a case in my "Lectures on the Ophthalmoscope."[2]

The accompanying wood-cut, Fig. 1, is an exact drawing from the preparation itself. The retina (*R*) is somewhat detached from the choroid (*Ch*). *V.r.* is a transverse section through the retinal vessels, and *V.c.* indicates the central artery and vein.

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[1] *v. Graefe's Archiv.*, Bd. iii., 1, S. 92.

[2] Berlin, 1864.

Fig. 1 is an illustration of how in atrophic conditions of the nerve the retinal vessels may disappear at the margin of an excavation. As opposed to a diagnosis of glaucoma in such an instance is the position of the lamina cribrosa—reaching to the choroidal plane; in an excavation due to pressure it lies considerably deeper than the latter. It follows from this that the larger the pre-existing physiological excavation the closer is its resemblance to the glaucomatous cup should the former become the seat of atrophy.

The lighter color of an atrophic optic nerve has an important bearing upon this question of pressure-excavation. Whether the atrophy is caused by the pressure-excavation or whether it has simply developed in a previously normal excavation can hardly be settled by the ophthalmoscope alone, for in the latter case the marginal line of the excavation may coincide exactly with the scleral border of the nerve and so present a "typical" picture of an excavation due to intraocular pressure, and yet be actually a result of optic atrophy without glaucoma.

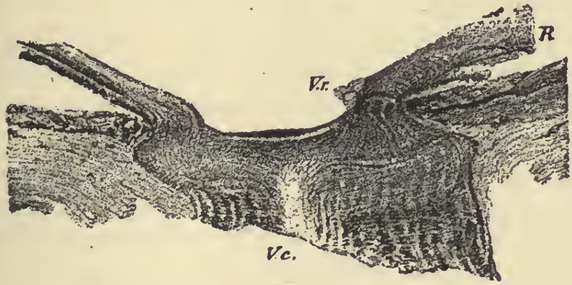

FIG. 1.

This confusion of optic-nerve affections with simple glaucoma is as old as the word itself. As an example of this see "Contributions to the Study of Glaucoma,"[1] in which a case of "glaucoma simplex" is described that produced blindness in thirteen weeks. The state of the visual field, but still more the clinical history, are opposed to the idea of glaucoma; the latter disease does not go on to complete loss of sight in so short a time, although some optic-nerve diseases may. So far as plus tension is concerned, there is no difficulty in discovering that, if one only has sufficient faith! I am convinced that all cases described as glaucoma simplex fulminans are really instances of atrophic degeneration of the optic nerve occurring with physiological cupping of the disk.

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[1] *L. c.*, Bd. viii., 2, S. 145.

If this be true there is no such thing as a *typical* excavation due to intraocular pressure, and when the character of the excavation is in doubt (especially in bilateral cases) it is not to be determined by testing the tension merely; all the other symptoms of glaucoma must be considered. The so called "glaucoma simplex" may thus be divided into two groups: first, true glaucoma, unaccompanied by marked inflammatory signs; and, second, a diseased optic nerve with a pre-existing physiological excavation. In what respect do these two groups differ from each other? I have already (in 1877[1]) drawn attention to the importance of this question, but shall now proceed to consider it more closely.

If one find in the papilla of one eye a marked excavation while the disk surface of the other is flat, it is certainly a case of glaucoma, because that is the only disease capable of excavating the disk. When, however, it is discovered in both eyes the diagnosis is more difficult, chiefly because optic-nerve atrophy may be the result of not one but of many quite different affections. Von Graefe has already cautioned us against the loose application of the term "optic-nerve atrophy" still in vogue among us, and has pointed out the importance of differentiating between atrophic degenerations and primary atrophy of the nerve fibres. Secondary atrophic degeneration may result from all those diseases which interfere with light-conduction in the retina or optic nerve, *e. g.* choroiditis, retinal hemorrhage, neuro-retinitis, retrobulbar neuritis, retinal embolism, etc.

Still easier is it to mistake a physiological excavation for glaucoma when the former is associated with certain forms of primary atrophy. The commonest variety, spinal atrophy, presents such characteristic signs that it is not likely to mislead, but the more uncommon form, that unconnected with spinal disease, may very easily be mistaken for simple glaucoma in the presence of a physiological excavation of the papilla. It is consequently advisable to watch the course of such cases, particularly in eyes that present no excavation of the disk. Sometimes their progress, just as in glaucoma simplex, is a very chronic one. I will, as an example, give the following history of such a case with a physiological excavation, which might very easily have been mistaken for glaucoma simplex:

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[1] "Ueber Glaucom." Clinical lectures published by R. Volkmann, No. 124, p. 1043.

CASE I.—R. S. æt. 76, in 1876 had u. o. V = $\frac{6}{9}$. Fundus normal then and after repeated examinations in 1878 and 1880. In April, 1881, V = $\frac{5}{12}$—$\frac{5}{9}$. O D a light gray. In October, 1885, V L=fingers with difficulty; a defect in the lower visual field; light reaction normal but O D white. V R = $\frac{5}{18}$; O D almost as white as L. In June, 1886, V L = hand movements. The atrophy had produced retraction of the papillary surface and a slight incurvation of the vessels was noticed at its border. Further loss of vision followed until death in 1888. Period from the commencement of the optic-nerve disease until complete blindness set in from six to seven years.

In another similar case, that ran its course to total blindness in eleven years, glaucoma was excluded by the early discovery of disturbed color-sense, while in the following case the mistake was actually made.

CASE 2.—von B., in 1882 had V = $\frac{1}{2}$ - 1. Apart from some peripheral lenticular opacities his eyes were normal. A small pit in the centre of the O D. In June, 1884, a slight discoloration of the papilla was noticeable. V R and L = $\frac{5}{9}$. December, 1885, left upper hemiopic defect in F of V following a slight attack of apoplexy. Both papillæ gray and showing a slight incurvation of the vessels at the medial border of the nerve. December, 1886, V R = $\frac{1}{3}$ - $\frac{1}{2}$, V L = $\frac{1}{4}$. This deterioration of V in the left eye showed itself after a sclerotomy had been perpetrated upon it, resulting in astigmatism visible by the keratoscope. In November, 1888, V R = $\frac{5}{12}$; V L = $\frac{5}{36}$ - $\frac{5}{124}$; O D as before, grayish-white. Patient died in 1889 of mental disease.

It is worthy of notice that the senile decoloration of the disk was so pronounced in this case that it was mistaken for an excavation; that there was no atrophic degeneration present is shown by the fact that for four years there was little or no loss of vision, except that due to the operation.

I have, of course, seen cases in which atrophic degeneration has been mistaken for an excavation, and by taking a step farther, one might, as C. Stedman Bull[1] has done, designate cases without excavation glaucoma simplex simply on the ground of deteriorated vision and a contracted field.

Finally, that atrophic degeneration of the nerve does not necessarily lead to blindness, but may remain stationary with an extensive defect in the visual field, is proved by the following history.

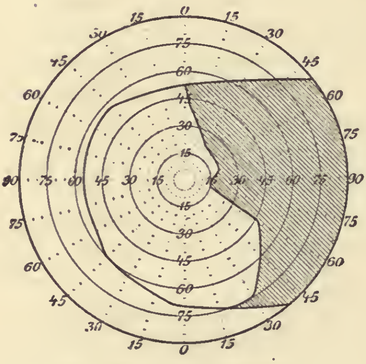

FIG. 2.

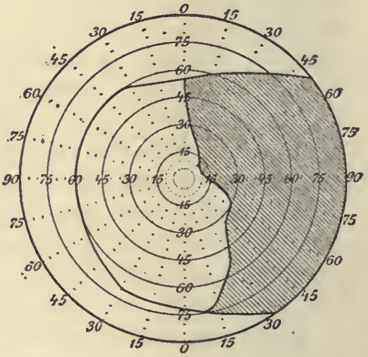

FIG. 3.

CASE 3.—Mr. M., æt. 50, consulted me about his presbyopia in December, 1874. Both eyes emmetropic and V = $\frac{10}{12}$. July, 1875, sight unchanged; V L = $\frac{10}{30}$; outwards a large scotoma, color-sense and pupillary reaction normal; O D somewhat "off" color. The vision in this eye grew gradually worse until, at the end of 1876, complete blindness resulted. The R E continued normal until April, 1879, when a small outward scotoma and a slight discoloration of the disk were discovered. Fig. 2 shows the F of V in May, 1879. Patient having had syphilis 20–25 years before, mercurial inunction was employed, with complete arrest of the disease. When last examined, January, 1888: V R = $\frac{3}{9}$; the F of V was as in Fig. 3; and the O D slightly paler than normal.

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[1] *N. Y. Med. Jour.*, Aug. 10, 1889.

The inunction treatment may have saved this patient, but almost every case of atrophic degeneration of the opticus free from spinal disease has been treated by me in this way without benefit. It is generally conceded that in the spinal form it is also valueless.

Since all these cases were complicated by the presence of physiological excavation they were doubtless difficult to diagnose from simple glaucoma. That I may lay additional stress upon the importance of diagnosis I report the following case of "typical glaucomatous excavation" *without* glaucoma, that ran its slow course to final blindness.

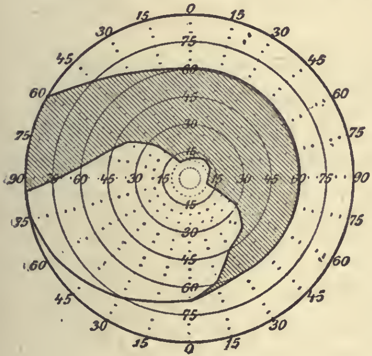

FIG. 4.

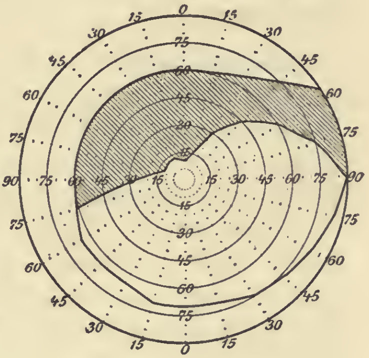

FIG. 5.

CASE 4.—Miss G., æt. 66, consulted me because some one had frightened her about her "excavation." V R + 1.25 = $\frac{6}{12}$; V L + 1.25 = $\frac{5}{9}$. Deep bilateral excavations reaching to the margin, but F of V uncontracted. Cornea clear; pupil normal; T + ? No halos or cloudy vision. No trace of glaucoma or excuse for iridectomy. In May, 1888, patient returned, after consulting another oculist, with vision decidedly worse. L V + 1.25 = $\frac{5}{24}$; scotoma up and in; V R + 1.25 = $\frac{5}{12}$; also a defect in the F of V (Figs. 4 and 5). Color-sense, cornea, and pupil normal. Papilla pale. Although I saw no reason to modify my previous diagnosis, I felt obliged to do an iridectomy. The case was growing worse, and as the patient had been urged to undergo the operation the increasing blindness would naturally be attributed by every future observer to my neglect. However, vision gradually sank until about a year after the iridectomy. $V\ L + 1.25 = \frac{1}{9}$; $V\ R + 1.25 = \frac{1}{5}$. In November, 1889, the question of glaucoma was decided, so far as the left eye was concerned, by numerous retinal hemorrhages. V = fingers at 20 *cm ;* $V\ R + 1.25 = \frac{5}{24}$. The scotoma gradually approached the fixation point. In the right eye marked tortuosity of the small retinal vessels. This is plainly marked at the bottom of the excavation —an observation I have made in similar cases of physiological excavation accompanied by atrophy, and consequently not to be regarded as characteristic of glaucoma. The F of V in the right eye contracted still more, and vision was finally lost from retinal hemorrhage.

From the beginning the term glaucoma simplex has not been precisely defined.[1] "Every oculist has certainly seen cases of excavated papillæ with increasing hardness of the globe, etc." "The increased tension from fluid pressure persists and becomes so great as to bring about atrophy of the nerve at the papilla, contraction of the arteries, and finally total blindness." This is said to be a picture of typical glaucoma without complication. I deny it. I also doubt the statement made on pages 151 and 155 that in simple glaucoma the eye may assume a stony hardness. On page 155 it is said that "inflammatory symptoms, as evidenced by hemorrhage, pain, and cloudiness of the media, often set in during the early stage of the disease." That is to say simple and inflammatory glaucoma may exist in the same eye.

This authority seeks to explain (page 150) von Graefe's initial stage of simple glaucoma by regarding it as the glaucoma itself. What meaning is attached to the term glaucoma simplex? Possibly the intermediate stages when the eye is absolutely normal and without glaucomatous symptoms, or does simple glaucoma begin with an initial stage of cloudy vision, halos, etc.? The term "initial stage" is an unfortunate one, as slight attacks are not necessarily the forerunner of more severe ones; gradually and without the latter the eye may slowly advance to blindness.

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[1] *v. Graefe's Arch.*, Bd. viii., 2, S. 143.

These slight passing attacks of glaucoma controvert the retention theory of the disease. How can one reconcile with this theory the fact that the symptoms set in so abruptly and disappear so quickly? In what way does the filtration angle become so quickly blocked up and as suddenly restored? Swelling of the ciliary processes has been held responsible for it. That inflammatory alterations may be discovered in eyes enucleated on account of painful, severe, and long-continued inflammation is self-evident. The ciliary processes in these cases may be atrophied or they may be swollen, but it does not necessarily follow that glaucoma begins with ciliary swelling. One of the few instances in which a recent case of glaucoma was examined anatomically is reported by Birnbacher.[1] The eye was examined seven days after the disease—acute inflammatory glaucoma—showed itself. The ciliary processes were greatly developed, but not to a greater extent than is often seen in normal eyes. There was neither swelling nor softening of the ciliary tissues; the blood-vessels presented no sign of parietal changes or of congestion.

Finally the greatest individual differences exist as to size between ciliary bodies. Fuchs[2] has described a case of glaucoma operated upon seven years before and with good results, in which after the death of the patient an uncommonly well developed ciliary body was found. Inasmuch as the eye had been free of glaucomatous symptoms for seven years, the extreme ciliary development cannot be regarded as the cause of the original disease. Otherwise one could hardly be asked to believe that with the cause continually active the effect should be entirely removed.

An obstruction at the filtration angle produced by ciliary enlargement would, in the beginning at least, result in an increase of aqueous and a deepening of the anterior chamber—a state of things which has never been observed. As everybody knows, the shallow anterior chamber of glaucoma is produced by a crowding forward of the crystalline and its attachments consequent upon an increase in the volume of the vitreous. In Birnbacher's report not only is this stated to be the case, but owing to tension upon the zonula the ciliary processes were dragged forward to the posterior surface of the iris, and in places came into actual contact with it. This, however, is not the cause, but simply the effect of increased intraocular pressure. The occurrence of glaucoma in aphakic eyes also bears witness against the retention theory. Of course, glaucoma rarely follows aphakia, but then aphakic eyes are much less numerous than those that are supplied with lenses. Moreover, a deep anterior chamber with aphakia is hardly compatible with the idea of an obstructed filtration angle due to swelling of the ciliary processes.

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[1] "Beitrag zur Anatomie des Glaucoma acutum." Graz, 1890.

[2] *v. Graefe's Archiv*, Bd. xxx., 3, S. 123.

Probably the chief and most frequent symptom of glaucoma is misty vision and the appearance of halos. These symptoms must not be *suggested* to the patient. Almost every one with weak sight complains of cloudy vision, and even "rainbows" may after a number of observations be seen, which are, however, often merely sensory hallucinations. Yet these statements must never be lightly regarded, especially when glaucoma is already present in one eye. That true halo-vision does not always indicate glaucoma is well known. The thin film of mucus that sometimes overspreads the cornea in cases of chronic conjunctivitis gives rise to these symptoms.

In examining cases of glaucoma in which halos have been seen, the observer will notice, along with conjunctival hyperæmia and increased tension, a slight cloudiness of the cornea. It is consequently a true diffractive effect, such as one obtains by looking through a misty window-pane, or by gazing at a light through a glass sprinkled with lycopodium powder. It is only in cases of slight cloudiness of the cornea that this appearance is seen; when the opacity is so marked as to interfere with an ophthalmoscopic examination, it is not perceived by the patient. That this symptom is rarely noticed by the patient is due to the fact that, as a rule, patients are not acute observers. For example, *in mild* *attacks of iritis with slight deterioration (less than about $\frac{1}{6}$) of vision, halo-vision may readily be observed if the sound eye be covered and the patient be told to look at a lamp that has been turned down low.* Not infrequently patients tell me of this appearance, and in cases like the foregoing it can readily be induced. The same is true of glaucoma, and if we regard the cloudy cornea in iritis as an inflammatory change, we must attach a like significance to it in glaucoma. In both cases it is an inflammatory œdema of the cornea. In this, as in other conditions, there are marked individual differences. For instance, it not infrequently happens that with slight hyperæmia of the conjunctiva, without pain, with a moderate degree of tension, and with an active pupil, the cornea may be so cloudy that the fundus is barely visible.

Finally, there is nothing characteristic about the corneal opacity; it is precisely the same in kind as that which accompanies iritis and irido-choroiditis. In connection with this occurs the question of a cloudy aqueous in glaucoma; but how can one distinguish through a cloudy cornea a turbid aqueous humor? The assertion that sometimes immediately after the escape of aqueous following iridectomy the pupil appears clear, proves nothing unless the cornea is examined by focal illumination, which in many cases is alone capable of indicating a slight but evident haze of the cornea. Furthermore, it is a rule that the corneal cloudiness only gradually disappears after iridectomy. The same thing may be said of the oft-quoted vitreous haziness. Glaucoma and a cloudy vitreous are not of necessity mutually exclusive, but the latter can only be demonstrated through a transparent cornea.

Another very important sign is the condition of the pupil. Glaucoma may progress to complete loss of sight without interference with the mobility of the iris. In many cases of unilateral blindness from glaucoma the direct reaction to light, of course, fails, but the convergent consensual-contraction remains.

Of the greatest diagnostic value is the dilatation of the pupil in glaucoma with inflammatory symptoms. In a glaucomatous attack, if the pupil be enlarged, we shall also find a cloudy cornea and increased tension, if the latter has not already passed away.

It rarely happens that the pupil is enlarged and at the same time round and perfectly movable. When one eye is completely blinded by glaucoma, such a condition might be induced if the other normal eye were accidentally covered. Avoiding this source of error I have, both in unilateral blindness from glaucoma, as well as in the earlier stages (with slight inflammatory symptoms), seldom found the pupil even moderately dilated and at the same time round and completely movable.

In most cases this enlargement of the pupil may be regarded merely as a ciliary paralysis from pressure, and as in other instances of this sort may be temporary or permanent. That the ciliary nerves are very sensitive to pressure is proved by the occurrence of *mydriasis traumatica*, which may follow contusions of the eye that leave behind them no other bad effect. It would be most unfortunate if the other motor nerves were as subject to traumatic paresis as the ciliary. In glaucoma paralysis of the ciliary nerves may be produced by one attack of sharply increased tension, but such a paralysis is not always complete. Sometimes the pupil is dilated in one direction only—most frequently upwards,—while in the remainder of the circumference the pupil still reacts to light, showing that some of the nerve fibres still retain their conducting power. Whenever we find the pupil fixed and widely dilated we may feel sure that the eye has been subjected to severe attacks of increased tension. Of course the presence of an inactive pupil does not necessarily imply glaucoma, but I have seen cases of atrophic degeneration of the optic nerve with immobility of the iris due to disease of the nerve centres diagnosed as glaucoma.

What has been said of the dilated pupil applies equally to the shallow anterior chamber. It is produced by a sudden increase of intraocular pressure accompanied by inflammatory symptoms due to enlargement of the vitreous body. The anterior space is again restored when the abnormal tension disappears. The diagnostic value of this sign is small.

Another symptom directly arising from this condition of increased tension is arterial pulsation, whose importance in the diagnosis of glaucoma von Graefe has already drawn attention to. Of course I refer only to the pathological form of arterial pulsation. A physiological increase of pressure in the retinal arteries follows the cardiac diastole, but its effects are not visible; only under certain pathological conditions, such as in affections of the heart and large vessels, Basedow's disease,[1] etc., can the systolic expansion and movement of the arteries be seen.

The arterial pulse, which in glaucoma can always be induced by pressure on the globe, is a decidedly abnormal occurrence. It may be recognized by the fact that *during* the heart's diastole the central artery is in places quite empty of blood. This arterial pulsation is the result of a disproportion between the intravascular pressure in the arteria centralis and the vitreous pressure. During the diastole the pressure of the vitreous preponderates and compresses the arterial walls at their weakest point—just where the vessel leaves the nerve trunk and makes a rectangular bend. That part of the artery thus emptied of blood commonly reaches to the margin of the disk, rarely past it. During the systole the blood pressure is in excess and the emptied vessel again becomes filled.

Usually the cause of the phenomenon lies in increased intraocular tension, but this is not always the case, since whatever reduces arterial tension may produce the same result. For instance, von Graefe[2] observed two examples of arterial pulsation in orbital tumors accompanied by neuroretinitis. It is possible that the tumor may have exerted on the eye an effect similar to finger pressure, or it may have compressed the ophthalmic artery and in this way reduced the intravascular tension. In a third case, of neuritis, it was probably produced by the space-reduction within the scleral ring—a result of the neuritis itself. Exactly the same condition may be induced by a general reduction of the blood-pressure as an expression of cardiac weakness. In a case of syncope B. Wordsworth[1] observed the arterial pulse set in immediately before the collapse of the patient.

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[1] Quincke, *Berlin. klin. Wochenschr.*, 1886, No. 34, and 1870, No 21. Also O. Becker, *v. Graefe's Archiv*, Bd. xviii., 1, S. 206, and *Wiener med. Wochenschr.*, 1875.

[2] *v. Graefe's Arch.*, Bd. xii., 2, S. 131.

Thus pulsation of the central artery can be produced even when the intravascular tension is normal if the intraocular tension be raised; it is also occasioned by the normal ocular tension when the arterial pressure sinks sufficiently low. It follows from this that in glaucoma the occurrence of arterial pulsation does not necessarily imply a marked increase of intraocular pressure. *I consider arterial pulsation alone to be insufficient evidence of glaucoma.*

Doubtless if a clear view of the fundus were not so frequently prevented by a cloudy cornea we would be able to discover in glaucoma more cases of arterial pulsation. Of greater importance is it to detect it in glaucoma without concomitant inflammatory conditions, even without the usual results of increased tension, such as dilatation and immobility of the pupil. I have repeatedly observed it when the pupil was normal and active, when the vision was normal, and when the tension was slightly increased or doubtful. I have often seen the central artery pulsate in such cases after simple movement of the eye.

That one can induce arterial pulsations by pressure of the finger upon the normal eye has been observed by von Graefe[2] from experiments on his own eye. "It is extremely interesting but somewhat dangerous to observe the arterial pulse in one's own eye after one has induced 'Purkinje's figures.' It seems to me that one can in this way follow the pulsation quite a distance from the blind spot. The very strong pressure, however, produces such severe and lasting symptoms that I would not advise any one to try it on his own eyes."

Of special significance is arterial pulsation in connection with retinal hemorrhage following iridectomy. When the anterior chamber is emptied of its contents the intraocular pressure instantly falls and blood rushes into the retinal vessels. As Cohnheim[3] has shown, this is a condition that favors hemorrhage; he has observed that the capillaries, for a time shut off from the circulation, are liable to rupture when the blood stream is again turned into them.

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[1] Ophth. Hosp. Reports, vol. iv., p. 111.

[2] *v. Graefe's Arch.*, Bd. i., 1, 8, 387–688.

[3] "Untersuchungen aus dem embolischen Process," 1872, S. 39.

Of course arterial pulsation is not a necessary forerunner of these bleedings. I have seen them take place in cases that just before the operation showed no sign of pulsation. This is also true of bleeding into the anterior chamber. It often occurs along with retinal hemorrhages, but the latter is such a common accident that it is not easy to establish a causal relation between them. It is worthy of remark that hemorrhage from both iris and retina may follow iridectomy for glaucoma in cases presenting few inflammatory symptoms.

But the most important alteration that accompanies glaucoma is that which the optic nerve suffers. It must, however, be remembered that there is but one way in which a correct estimate of the clinical meaning to be attached to a pressure-cup can be reached, and that is by comparing it with an earlier picture of the papilla. *Furthermore, I may state that glaucoma may proceed to final blindness without the formation of an excavation.* The following is a case in point.

Case 5.—Mrs. F., æt. 54, came under treatment the beginning of December, 1878. Two years before said to have had severe inflammation of *right* eye with headache. Three months ago another painful inflammatory attack in the same eye, which resulted, after about eight days, in complete blindness. The *left* eye was said to have had good vision until eight days ago, when she had a severe attack of inflammation accompanied by nausea and vomiting. Vision is almost entirely destroyed. Only inwards and upwards are hand movements still perceived. There is a slight degree of tension in both eyes—more in the right than in the left. Bilateral pericorneal injection; cornea cloudy, the central area most affected; anterior chamber not encroached upon; red reflex indistinct; eserine produces but slight effect.

December 3, 1878 (under chloroform) T ?; sclerotomy in both eyes. December 13th, right cornea transparent; optic nerve excavated; no retinal hemorrhages; *left* cornea transparent; *no excavation of papilla, which appeared quite normal.* Vision finally reduced to perception of light and the uncertain discernment of large objects in eccentric portions of the field.

It is worthy of note that a *single* attack of glaucoma can induce blindness in an otherwise healthy eye. When the patient above referred to applied for treatment, eight days after the attack, the increase of tension was fast disappearing. The optic nerve, visible after the operation, seemed quite normal, and, although later on atrophic degeneration showed itself, there was no excavation.

In addition to this, large defects in the field of vision may be developed without excavation.

Case 6.—Mrs. L., æt. 36. January 23, 1883 : *Right*, sharp attack of inflammatory glaucoma for past eight days ; cloudiness of cornea and mydriasis ; V = hand movements only ; defect of medial half of field. Iridectomy, January 24th. Evening of January 25th, *left*, slightly cloudy cornea and dilated pupil. In the night a severe attack. January 26th, iridectomy. February 13th : R V = fingers at 1 metre. O D not excavated but lighter in color. November 17th : V R = $\frac{1}{18}$; O D not excavated but white ; V L + 1 = $\frac{1}{2}$; O D normal.

Case 7.—Florentine L., æt., 49. March 27, 1882 : Glaucoma in *right* since November. V = fingers at 1 *m*. Contracted F of V ; mydriasis and haze of cornea ; fundus not visible ; + T ? ; slight effects from eserine ; V L + 4 = $\frac{6}{18}$; previous convergent strabismus. March 28, 1883 : *Right*, iridectomy. April 7th : Still some blood in anterior chamber ; no excavation ; O D somewhat paler than left ; some small retinal hemorrhages at the equator outwards ; V + 3 = $\frac{1}{12}$ + 6. No. 0.3 slowly ; scotoma above and in.

Fig. 6 is the perimeter chart, measured March 27th. It is not an exact one, as might be expected from the low vision. Fig. 7 shows the F of V projected on a flat surface ; the medial scotoma corresponds in form to the perimetric measurement ; the temporal half of the field, on the other hand, was normal.

In all these cases, if there had been a physiological cup, a marked excavation from pressure would have been seen, but this would not have been the cause of the blindness, inasmuch as the latter resulted from pressure-paresis of the retina or the optic nerve. At first the papilla looked quite normal, and it was only later on that atrophy showed itself as a result of the blindness, just as, for instance, we find it following destruction of the opticus fibres from hemorrhage into the retina. Of course such a paralysis due to pressure presupposes a sharp increase of the intraocular tension which only occurs in severe attacks of glaucoma. In those cases that pursue a chronic course, the formation of an excavation is a gradual process, and when a physiological pit already exists it affects like it the centre of the disk. I have seen cases, in which there was no physiological pit, after the arrest of the disease by iridectomy, present an excavation that, in so far as it did not extend to the papillary margin, closely resembled the normal condition. In the earlier stages of the pressure-cup the atrophic discoloration is commonly absent, but when the glaucoma is of long standing it can always be seen. *The ophthalmoscopic picture of a physiological cup may be produced by glaucoma, just as extensive normal excavations with discoloration of the disk cannot be distinguished from excavations due to intraocular pressure.* Small physiological excavations (which affect about one third of the disk surface) are quite common, and the larger the normal cup the sooner it enlarges under the influence of pressure and reaches the border of the papilla. All this once

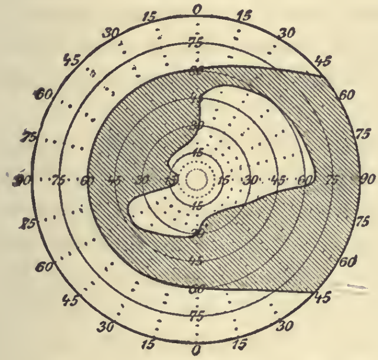

FIG. 6.

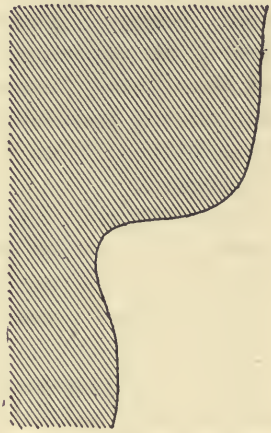

FIG. 7.

more teaches us not to allow the diagnosis of glaucoma to rest upon a simple symptom, but to take into consideration every clinical feature. The length of time occupied by glaucoma in the excavation of a level disk depends upon a variety of circumstances. It is not uncommon to run across cases that have persisted for years, having a history of attacks accompanied by cloudy vision and halos, until a more severe attack than usual called for an operation, and yet no pressure excavation resulted. The occurrence of this sign is evidently governed by the amount of the intraocular pressure, the duration and frequency of the several attacks, and the power of resistance inherent in the nervous tissue itself. Finally, a disk with even a small excavation sooner presents the appearance of having been excavated by pressure than does a flat papilla with little or no central pitting. As far as my observation goes, the excavation of a level disk proceeds for at least six months before it reaches the papillary border.

A true pressure-excavation, so affected that the lamina cribrosa is gradually pressed backwards, is always accompanied by an interruption of the nervous conduction. This is evidenced ophthalmoscopically by a discoloration of the excavation floor, and functionally by absolute scotomata. As is well known, the medial (inner) half of the visual field is first affected. Although I believe it possible that central defects may also result from pressure, I have had no experience of them. It is, however, important from a diagnostic standpoint to remember that every true pressure-excavation implies a defect of the visual field ; conversely, even when the excavation encroaches upon the border of the disk and there is no scotoma, the pitting is not due to intraocular pressure.

In my opinion, the diagnostic value of the *halo glaucomatosus* is quite as small. It consists of an annular discoloration of the choroid surrounding the papilla, such as is quite often seen in normal eyes. I am convinced, as the result of an anatomical examination,[1] that the pale narrow ring forming the border of the glaucomatous pit results from a complete atrophy of the choroidal circle that immediately surrounds the papilla. I found the choroid in this situation converted into a very thin, completely transparent membrane, such as may be seen in the advanced atrophy of axial myopia. There is, however, this difference : in extreme myopia the atrophic areas shade off into neighboring and less affected parts, while the glaucomatous ring stands out in sharp contrast with the normal choroid surrounding it. This atrophy in the case of glaucoma may be explained by the fact that sometimes the filaments forming part of the choroidal ring bury themselves in the lamina cribrosa, and when under the influence of increased tension the latter is pressed backwards the fibres are put upon the stretch, and atrophy results. The form of the atrophic area is seldom exactly annular, but is often irregularly indented towards the periphery, both by the encroachment of normal pigment as well as by the changes incident to axial myopia. It sometimes happens that the choroidal ring surrounding a glaucomatous excavation is deficient, so that the excavation appears as if surrounded by the sclerotic in the shape of a narrow, ring-like border. Nevertheless, the *halo glaucomatosus* may be absent about pressure-excavations, or it may be present unaccompanied by glaucoma.

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[1] "Vorlesungen über den Augenspiegel," Berlin, 1864, S. 131.

Of those cases of optic-nerve excavation which are commonly called simple glaucoma, I have classified a fair number as distinctly glaucomatous. These are the cases that present a deep *unilateral* pit reaching to the margin of the disk. Inasmuch as the larger physiological cups are always bilateral, we may with certainty consider deep, one-sided excavations to be the result of increased intraocular pressure. Furthermore, into this class should be admitted only those cases that have normal iris movements—*i. e.*, distinct convergence-contraction, or, if the eye be blind, consensual contraction to light. If the iris in such cases of unilateral excavation be dilated and immovable, it is an indication of previous inflammatory attacks. This is true even when at the time of the examination no other traces of these attacks can be discovered, because outbreaks of glaucoma followed by paralysis of the sphincter iridis are always inflammatory in character. This classification as glaucomatous of cases occurring without visible signs of inflammation (or at least of cases in which the diagnosis of a previous inflammation is merely inferred from present conditions), gives rise to a query whose answer must first of all be considered. The question relates to the development of glaucoma, and the conclusions can, of course, only be justified by the history which the patients themselves furnish. It is to be expected that the larger number have experienced the so-called initial symptoms, occasional halos, cloudiness of the visual field, etc., and many have had pains in the eye and over the forehead. The majority have at least had a variety of symptoms, while persons who are so innocent as to get blind in one eye without having meantime noticed anything remarkable, or who have discovered their blindness by accident or on being examined, can hardly be regarded as trustworthy in the presence of more reliable testimony. Finally, that these symptoms may be easily overlooked is proved by the fact that the halo-vision so common in iritis is rarely noticed by the patient. All patients affected by the so-called glaucoma simplex who had noticed their own condition have also remarked the occurrence of symptoms. *Whence I conclude that the course of the disease is marked by distinct attacks of increased tension that come on and (in the early stages at least) again subside.*

Whether these paroxysmal attacks are associated with inflammatory symptoms depends upon the particular case, just as in other ocular inflammations observation teaches us that one and the same pathological condition may be marked at one time by severe and at another time by very slight external signs of inflammation.

We may feel sure that the same intraocular conditions obtain in glaucoma as in irido-choroiditis, and that they vary in individual cases according to circumstances, and that the symptoms may be severe in some instances and barely noticeable in others. *The most important consideration is that the inflammatory symptoms of glaucoma are not the cause but the results of increased tension.* The rapidity with which it develops is more significant than the degree of intraocular tension. Considerable increase of tension may be present without glaucomatous inflammation, but a sudden rise of intraocular pressure is a cause of inflammatory symptoms. As a proof of this, I quote the following case.[1]

K. G., æt. 66, was, January 5, 1865, admitted to the eye clinic of the Vienna University on account of acute glaucoma in her left eye, on which iridectomy was performed the same day. The patient thought her right eye was sound, but on examination tension was slightly raised. The a. c. was very shallow, and the retinal veins were markedly congested and tortuous. On the evening of the fifteenth day, after operation on the right eye, the patient was suddenly attacked by violent pains in the forehead and right temple, accompanied by a cloud of mist and showers of golden sparks before the right eye. Dr. Rydal found the eye free of ciliary injection, but remarkably hard. The pupil was dilated; media fairly clear; marked arterial pulsation; swelling and tortuosity of the veins; venous pulsation at the exit of the vessels; halo-vision; slight decrease of visual acuteness. There was an hourly increase of ciliary injection; œdematous swelling of lids and conjunctiva bulbi showed itself, and the media became more and more cloudy. Rest was obtained that night by a hypodermic injection of morphia. Next morning showed a completely developed attack of glaucoma; the cornea dull; the iris immovable; the pupil widely dilated; a high degree of tension; fundus examination impossible, owing to cloudy media. Iridectomy was immediately performed and recovery followed.

Cases of glaucoma that occur in the second and sound eye, while the healing process is still going on in the iridectomized eye, furnish us with an opportunity of confirming the foregoing observation. In Rydal's case it should be noticed that the increase of tension preceded the inflammatory symptoms, and was undoubtedly the cause of the latter. Instances in which the converse of this is true—where, although the intraocular pressure has disappeared, signs of inflammation persist—occur more frequently.

We may then conclude that so far as concerns those cases of glaucoma "simplex" that are really glaucomatous, but which at the time of the examination present no inflammatory signs, the careful observer will recognize in them the symptoms of the so-called "initial stage." The fact that nervous irritation, etc., may occasionally provoke glaucoma, proves that nervous influences may determine periodical attacks of increased tension. It is probable that in this lies the best explanation of the frequent occurrence, after an iridectomy for glaucoma, of the disease in the other eye. The ocular apparatus affords several examples of sympathetic secretion. A physiological instance is crying, a pathological one herpes frontalis. This limitation to sharply defined areas upon one side of the head is sufficient evidence that the outbreak of herpetic vesicles, as well as the inflammatory symptoms that accompany them, have a nervous origin. We know as little about their physiological connection as we do in the case of glaucoma.

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[1] Dr. Lucian Rydal: "Ueber Glaucom."—Bericht über die Augenklinik der Wiener Universität, 1863–65. Wien, 1867. Bei Braumüller.

The ciliary body is undoubtedly the locality in which the sympathetic secretion of glaucoma takes place. Here it is that liquids can most easily be poured into the vitreous body, and so increase its volume. It is possible that the first onset of glaucoma may be a very severe one and attended by disastrous results, but as a rule the disease is ushered in by a number of separate, mild, and transient attacks. The increase of intraocular pressure may, as observed in the so-called prodromal stage, be entirely transitory, but in the course of time it becomes a permanent symptom that may at all times be detected by comparison with the other eye. The picture of a glaucoma simplex with a slowly increasing intraocular tension, creeping on to blindness without variation in its signs and spmptoms, is a purely fanciful one. Of course no one can be prevented from believing in the existence of such cases, if he accepts as true a carelessly taken history, but the *proof* is still wanting.

The above-described slow and uninterrupted loss of vision in "glaucoma simplex" indicates, as might be expected, progressive optic-nerve atrophy with physiological cupping of the disk. The diagnosis is, of course, not always easy. Where one papilla shows a distinct excavation, while the other disk is flat, the case is one of glaucoma without further question. But in the presence of physiological excavations the matter is often extremely difficult to decide. I have seen cases in which the pitting was large and bilateral, but not of the same size. While on the one side there was a narrow interspace between the pit margin and the papillary border, the other presented an exact picture of the so-called pressure-excavation, but vision was normal and the visual field uncontracted. I can only conclude from this that *physiological excavations are always bilateral, but are not always identical in size and location.* Such cases as these call for extended and careful observation. If the tension does not increase, if sight does not deteriorate, if the field of vision remains of normal extent, we may exclude glaucoma —it matters not how many "typical" signs of the disease may be present. With every excavation from pressure there of necessity go visual disturbances as soon as optic atrophy has developed. Although central vision may continue to be normal for a long time, restriction of the visual field finally shows itself, and this is equally true when the physiological excavation enlarges under intraocular pressure. Still more obscure is the diagnosis when a physiological excavation is complicated by an optic-nerve disease. Glaucomatous contractions at first always affect medial (inner) sections of the field of vision, but this may also occur in atrophic degenerations of the optic nerve. The color-sense may be unaffected in glaucoma, but this, too, is the case in some optic-nerve affections; the differences may be just as small as that between physiological and pressure-excavations. There are examples of undoubted glaucomatous excavations that do not extend to the margin of the disk, which it is not possible to distinguish from physiological cupping. In bilateral disease the determination of the tension is an uncertain diagnostic method. If one eye appears to be abnormally hard while the other is of normal tension glaucoma, is to be suspected, but + tension in both eyes proves nothing, inasmuch as normal tension presents the greatest individual variety.

The only road to success in diagnosis lies in continued *observation.* First of all patients must be taught to note their own symptoms, especially whether they are subject to occasional attacks of cloudy or rainbow-like vision. If this is not practicable and excavation, accompanied by scotomata, is present, the question arises whether we should perform an iridectomy or not.

One cannot discuss the subject of iridectomy without thinking of von Graefe. How many thousands have been saved from impending blindness by his ingenious discovery! **As soon as the existence of glaucoma has been demonstrated iridectomy should be performed at the earliest opportunity.** One may temporize for a few days with eserine, etc., but, as a rule, the time so occupied is that much time lost. In some cases of glaucoma that come for treatment on account of inflammatory symptoms it is sufficient, as von Graefe[1] has mentioned, to excise a portion of the sphincter iridis. In proof of this I will cite the following case mentioned in von Graefe's article[2] on glaucoma:

CASE 8.—Mrs. Maas, 60 years old, was treated by me in 1875. In February, 1857, she had been operated upon by von Graefe, first in the right eye, and five days later in the left. The left eye, which developed inflammatory symptoms shortly after the operation on the right, recovered with good vision. Eighteen years afterwards (April 5, 1875) the condition of this eye was as follows: the iridectomy, made inwards and somewhat downwards, is very small, barely 2 *mm* wide; the corneal incision is unusually long. Probably on account of the shallowness of the anterior chamber the operator was obliged to enter the keratome parallel to the plane of the iris, and so push it forward at an acute angle with the posterior surface of the cornea. The inner edge of the wound lies some distance from the scleral margin and is adherent to the iris throughout its whole extent. Vision is greatly reduced on account of a nuclear cataract, which was extracted July 30th. After discission of a secondary cataract, November, 1875, V with $+ 12 \text{ D} = \frac{12}{30}$.

Although the patient lost her right eye some years later from optic-nerve atrophy, yet her visual acuteness continued to be good in spite of the anterior synechiæ, which must have prevented a restoration of the irido-scleral angle.

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[1] *v. Graefe's Archiv*, Bd. xv., 3, S. 114.

[2] *L. c.*, Bd. iii., 2, S. 514.

The operation is comparatively more effective in marked inflammatory cases than in those unaccompanied by signs of inflammation. In the former instance the visual disturbances are the result of several causes: (1) the corneal haze; (2) the pressure-paralysis of the retina; (3) when present, the optic-nerve excavation. The corneal opacity alone may reduce the vision to the counting of fingers held immediately in front of the eye. If, however, there be no restriction of the visual field, one may expect the patient will regain useful vision. In the absence of a cloudy cornea the reduction of visual power may be attributed to the optic-nerve excavation. Every excavation due to pressure is followed by contraction of the field of vision, although, of course, an optic-nerve affection with a physiological excavation is, as a rule, also accompanied by similar defects.

Von Graefe noticed [1] that the deterioration of vision following immediately after operation occurs in those cases of glaucoma whose scotoma stretches almost to the fixation point.

Astigmatism, due to the enlargement of the pupillary area (producing wider dispersion circles), as well as to the healing of the corneal wound, is to be held responsible for some interference with vision, and it is easy to understand how these effects are more detrimental in eccentric than in centric fixation. Even central vision is affected by both iridectomy and sclerotomy, yet this fact is not to be weighed against the lessened danger from blindness. These drawbacks are, of course, quite unconnected with the glaucoma; they have as important if not a much more important bearing on optic-nerve affections with physiological excavation of the disk.

I have quite frequently operated on cases of glaucoma having a very small field (about 10° radius) but with good central acuteness, and after the iridectomy there has been no further loss of visual power. It is otherwise with cases of optic-nerve atrophy with a normal excavation, as, of course, a progressive disease of the nerves is not to be checked by iridectomy.

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[1] *v. Graefe's Archiv*, Bd. xv., 3, S. 182.

In cases of glaucoma we may count with a probability approaching certainty upon a cure of the disease by iridectomy. Exceptions to this rule occur, but happily not very often. I have repeatedly seen cases presenting some glaucomatous symptoms even after a correctly performed operation.

CASE 9.—Mr. S., æt. 23, comes of a glaucomatous family. His father and uncle were operated upon by me for glaucoma. When first examined, February, 1883, patient reported

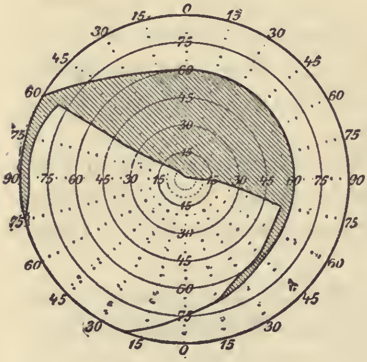

FIG. 8.

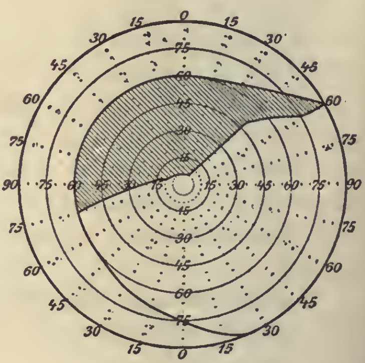

FIG. 9.

that he had had frequent attacks of rainbow-vision during the previous six months. V L = $\frac{5}{6}$; V R with − 0.75 = $\frac{5}{6}$; visual field not contracted ; bilateral, deep, and abrupt excavations ; angular bending of vessels at the papillary border. The halo-vision continued, and was unmistakably observed by the patient, who, as soon as he noticed anything wrong with his eye, was in the habit of striking a match to see whether or not there were any colored rings about the light. May 1, 1886, in both eyes V = $\frac{5}{6}$, but, in each, defects (see Figs. 8 and 9) in the upper half of the field, approaching the point of fixation. Cornea clear ; pupil not dilated ; the excavation (inverted image) reaches the upper margin of the nerve and thus corresponds completely with the situation of the scotoma ; in the right eye there is (indirect examination) a *halo glaucomatosus* at the upper border of the nerve ; the left eye in this situation shows only a slight atrophy of the pigment epithelium. Bilateral upward iridectomy. In spite of this the rainbow-vision before the right eye continued, and in November gave rise to considerable anxiety on account of its frequency. In the floor of the excavation, also, distinct pulsation. Owing to the displacement of the blood-vessels only one—the artery—can be seen. November 15th, a second and downward iridectomy was done in the right eye. An unusual degree of astigmatism was the result, the keratoscopic image presenting vertically an elongated oval. The halo-vision persisted for some time but gradually disappeared.

But these cases do not always terminate so satisfactorily.

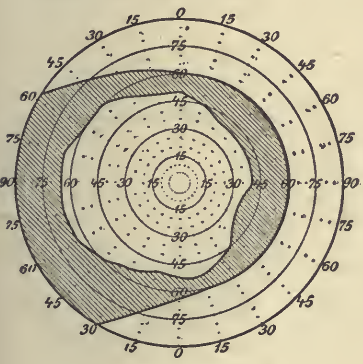

FIG. 10.

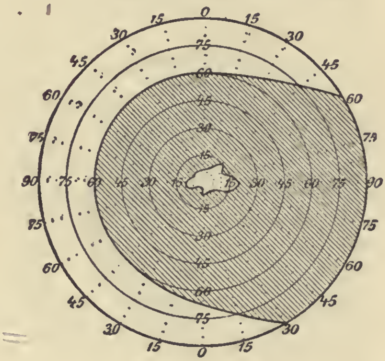

FIG. 11.

CASE 10. — W. T., æt. 46, treated October 20, 1886. Patient has had (right eye) cloudy, rainbow-vision for eighteen months. V R = $\frac{6}{18}$, + 0.75, 0.5 at 25 *cm.* Excavation reaching everywhere to border of disk; distinctly visible lamina cribrosa; sometimes spontaneous arterial pulsation. V L — 5 = $\frac{1}{12}$; 0.9 at 12 *cm.* Excavation to border of papilla. Broad peripheral iridectomy, both eyes. After this occasional arterial pulse and halo-vision. A year later the right eye became suddenly worse; pulsations in bottom of excavation; optic disk white; hand movements noticed only in a small portion of the field. April 16, 1890, spontaneous arterial pulsation, which again disappeared. Fig. 11 represents the right field of vision, taken before the iridectomy, October 20, 1886; Fig. 10, the left, April 17, 1890.

The following is a similar case:

Case 11.—Mr. H. had for years cloudy and halo-vision. In December, 1873, V R = $\frac{12}{30}$; V L = $\frac{12}{30}$; defect in visual field towards inner side; bilateral excavation extending to margin. Patient could not be persuaded to submit to an operation until October, 1875. Hazy vision and rainbows continued; V, both eyes, was $\frac{12}{30}$, but the right-sided scotoma had greatly increased in size, and in addition to it, on the left side, was another up and in. October 23d, iridectomy. Patient still frequently complained of cloudy vision, particularly in the morning. The visual acuteness of the left eye was preserved, but in the right it was lost. Figs. 12 and 13 give the visual fields taken March 26, 1876. June 28, 1878, V R = $\frac{5}{36}$; V L = $\frac{5}{24}$. January 4, 1887, V R = fingers (above and out only) at about ½ *m*; V L = $\frac{5}{18}$. April, 1891, the same.

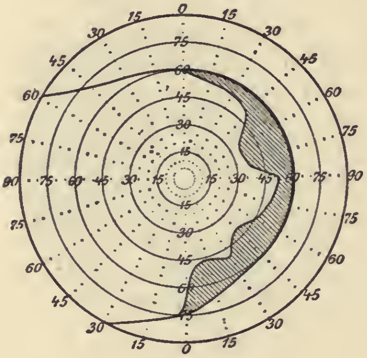

FIG. 12.

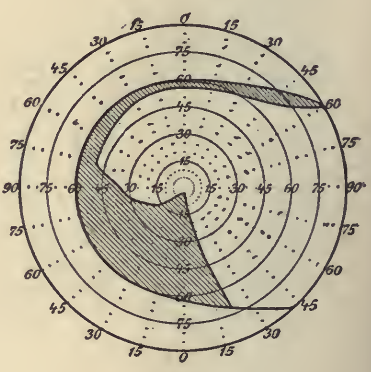

FIG. 13.

The statement that subsequent impairment of vision can be checked by repeated iridectomies or sclerotomies is not always justified by experience.

Although a malignant quality cannot be denied to those cases which do not exhibit the usual good results following iridectomy, the term *glaucoma malignum* is generally reserved for those that do not run a normal course after operation. On account of their rarity I consider it worth while to report those that have occurred in my private practice.

Case 12.—Mrs. H., about 50 years old. Condition July 12, 1871: V L (M $\frac{1}{50}$) = $\frac{1}{4}$; + $\frac{1}{10}$ J No. 10. Scotoma in and below; good eccentric vision only, out and above; marked increase of tension ; media clear ; pressure-excavation. R E — $\frac{1}{48}$; V = $\frac{10}{30}$; uncontracted field ; optic nerve normal ; no excavation, only a small natural pit. July 14th, on left eye a perfectly regular upward iridectomy. All went well until evening, then nocturnal pains, lachrymation, and injection of conjunctival sclera. Inflammation grew worse next day and developed a serious swelling of scleral conjunctiva, especially at inferior limbus, with a few conjunctival hemorrhages. On the pupillary margin of the discolored iris also a small hemorrhage. An empty anterior chamber and the eye harder than before the operation. After about four weeks the inflammatory symptoms subsided and the tension had diminished somewhat, but the a. c. continued empty, and during the later course of the disease the left was subjected to frequent inflammatory attacks. Last examination end of Nov., 1872 ; the a. c. in same condition ; cornea irregular through loss of epithelium. Lens reflex very marked ; ophthalmoscopic examination showed centre of pupillary area hazy, only the periphery transparent ; fundus not visible. Anterior perforating veins enlarged. The eye is still hard. No cystoid cicatrix, only a small transparent vesicle in the scar. Right, condition unchanged.

Case 13.—Mrs. O., æt. 60, a depressed, nervous individual. May, 1872, V R — $\frac{1}{36}$ = $\frac{10}{15}$; scotoma up and in ; commencing excavation ; T + ; frontal pains ; halo-vision ; V L — $\frac{1}{48}$ = $\frac{10}{12}$; normal field and no excavation. Oct. 28th, excavation almost complete. Nov. 13th, V = $\frac{12}{70}$; + $\frac{1}{16}$ J No. 5. Nov. 18th, puncture of a. c., which immediately filled. Nov. 21st, upward iridectomy, followed next day by pains, and later on by swelling of upper lid and symptoms as in foregoing case. Wound did not close until Dec. 15th. The a. c. still remained empty, although the media became clear. Vision reduced to perception of light in a small, external portion of field. Tension normal. Five years later : most of the right iris attached to the cornea ; anterior chamber very shallow or empty ; V L (M 1.5) = $\frac{5}{6}$; no excavation.

It is worthy of note in this case that although the paracentesis wound healed smoothly, the iridectomy resulted so disastrously.

Case 14.—Miss M., æt. 48, advanced case of glaucoma in left ; iridectomy followed by severe inflammatory reaction ; cystoid vesicles in scar and total loss of vision.

Case 15.—Mrs. L., æt. 65. July 2, 1872: right eye glaucoma absolutum. Thirteen years before von Graefe did an iridectomy on this eye, after which, according to patient's story, healing was slow and irregular. Left eye growing worse since Christmas, 1871. Glaucomatous symptoms not well marked; V = $\frac{10}{40}$. Iridectomy, July 12th, in good form. In eight days posterior synechiæ; later, hemorrhage in iris at pupillary border. Vision grew worse until, in the middle of August, it was reduced to finger-counting at five feet. Lens reflex; empty a. c.; eye hard. Last examination, Jan. 23, 1873: cystoid cicatrix; a. c. almost empty; nuclear cataract but equator lentis clear in iridectomy region; V = fingers at 1–2 feet.

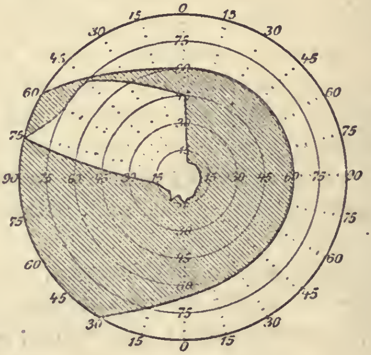

FIG. 14.

Case 16.—Miss F., aged 47. V L = 1. Right: commencement of blindness not noticed; temporary cloudiness; pains; no halos. Jan. 13, 1875, V = fingers uncertain and eccentrically; large scotoma; deep excavation; pupil not dilated and media clear; marked increase of tension. Jan. 19th, iridectomy. Eye still hard; much lachrymation and conjunctival hyperæmia. The a. c. remained empty. March 5th, vision reduced to bare perception of light. April 23, 1878, iris applied to cornea; eye atrophic; cataract. Left, still normal.

Case 17.—Mrs. W., aged 49. Aug. 3, 1876. Three years before, "burning" in right eye; gradual loss of sight; excavation with white background reaches to edge of disk, + T. *Left*, glaucoma with marked inflammatory symptoms. V = $\frac{1}{7}$; F of V restricted as in Fig. 14. Aug. 4th, iridectomy, both eyes. Iris angles well replaced. In spite of careful treatment and watching, both eyes went from bad to worse until Jan., 1877, when V L = hand movements ; a. c. empty ; cloudy cornea. March 19th, left, same vision ; right, no perception of light.

CASE 18.—Mrs. B., æt. 45. June 28, 1881 : left, for one half year, halos and cloudy vision ; $V = \frac{1}{3} - \frac{5}{12}$; large scotoma and excavation ; cornea clear ; no dilatation of pupil ; + T. Right : $V = \frac{5}{6}$; optic nerve and F of V normal. July 1st, iridectomy ; left, small anterior synechia, but angles free. Sept. 17th, V L = fingers at 1 *m* ; scar broad and dark-colored ; a. c. shallow. March 6, 1882, left, complicated cataract ; right, normal.

One case which I was at first disposed to regard as an example of glaucoma malignum I now consider to be of the same nature as those cases of irido-choroiditis terminating in blindness that in pre-antiseptic times (to which it as a matter of fact belonged) so frequently followed cataract operations.

The most tedious restoration of the anterior chamber is not necessarily evidence of glaucoma malignum since it is commonly encountered in cases where increased tension has persisted for a long period. The aqueous chamber may remain empty for six to eight days without evil consequences. Nor is its evacuation after iridectomy in glaucoma malignum the cause of the subsequent trouble. In Case 13, for example, where a paracentesis corneæ was done several days before the operation, the chamber immediately refilled, although it remained a long time empty after the subsequent iridectomy.

In addition to the foregoing cases of glaucoma malignum I have seen others not operated upon by me. From this varied experience I deduce the following rule : *When glaucoma malignum attacks one eye, it follows iridectomy on the other eye, even when the second eye is not affected for years after the first.* The cause of the unsatisfactory results of the operation must lie in some obscure structural anomaly inherent in the eye itself. *Experience teaches us that in all cases of chronic glaucoma affecting both eyes it is advisable to operate first of all upon the worse one, even if it be absolutely blind. Should this be followed by the normal-healing process the second eye may be operated upon without the least apprehension.*

Fortunately, cases of glaucoma malignum occur so rarely that special mention of them is made in glaucoma statistics. All my private cases presented themselves between 1871 and 1881, and up to the present time I have not had another.

In any event these rare cases of glaucoma malignum furnish no excuse for preferring sclerotomy to iridectomy. *We may count upon a favorable result following iridectomy with almost perfect certainty, while sclerotomy, on the other hand, is by no means an infallible remedy.* In support of this opinion I can furnish a number of examples.

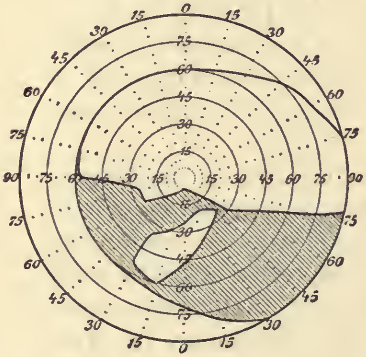

FIG. 15.

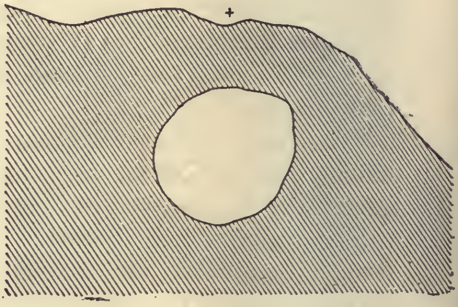

FIG. 16.

Case 19.—I. S., æt. 33, March 11, 1878, *left*, rainbows for six months; cornea clear; small pupil; V = $\frac{6}{9}$; uncontracted field. Right, V = $\frac{6}{12}$. Large physiological excavation of both disks. July 1, 1880, *left*, V = $\frac{5}{24}$, 0.6 slowly at 20 *cm*; + T; peculiar inferior scotoma that encloses an unaffected retinal area. This is represented in Figs. 15 and 16, the first a perimetric measurement, corresponding to a control projection (Fig. 16) on a plane surface. Halo-vision more frequent; cornea clear; pupil not dilated; R V = $\frac{5}{12}$, 0.24 at 20 *cm*; normal field; peripheral vision good. July 30, 1880; left, sclerotomy. May 24, 1881, left, V = peripheral (best outwards), finger counting. Right, V = $\frac{5}{18}$ – $\frac{5}{12}$; peripheral V good.

Sclerotomy did not influence the course of the glaucoma. Of course the subsequent iridectomy performed on the left eye did not effect an improvement.

CASE 20.—Mrs. St., æt. 64, July 18, 1876. A year ago a sudden cloud before the right eye, lasting several months. No reliable history; clear cornea; pupil not dilated and consensual movements normal, but no reaction to right; deep excavation; + 4 D, No. iv. Sn. at 10″. Small central F of V, contracted as per Fig. 18; + T. *Left:* V + 4 = $\frac{5}{12}$ – $\frac{5}{9}$; no excavation and *F of V unrestricted.* July 27, 1876, right, iridectomy. September 22, 1876, right + 6 = letters of Sn. vii. Left, + 4, V = $\frac{5}{12}$; + T. Slightly hazy vision; some pain in ocular region; no excavation. March 12, 1877: R + 6 = No. 4, 30 *cm*; V = $\frac{3}{40}$, L + 4 = $\frac{6}{6}$. February 18, 1881: Slight glaucomatous attack; *left, sclerotomy,* February 21st. March 10th: L + 4 = 0.4 at 15 *cm.* March 14th: cloudy vision and pain. April 17th: L V + 4 = $\frac{5}{12}$; R V = doubtful perception of light. July 15, 1882: left + 6 = 0.3 in 20 *cm*; beginning excavation.

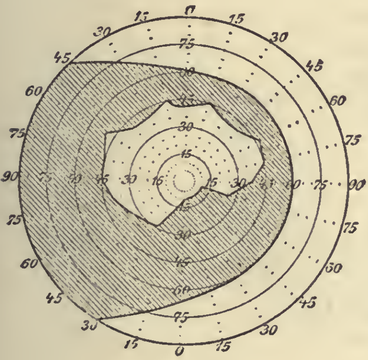

FIG. 17.

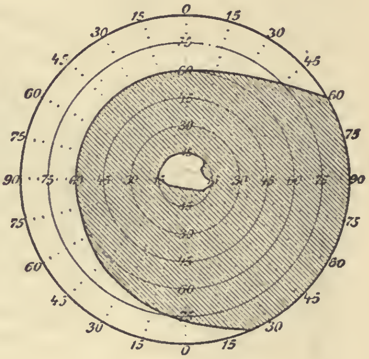

FIG. 18.

The left eye grew progressively worse until finally on December 24, 1883, an iridectomy was done. The visual field at that time was as shown in Fig. 17. May 24, 1885, V = $\frac{6}{36}$; beginning cataract.

The sclerotomy did not check the progress of the glaucoma, which, however, was accomplished two years later by an iridectomy.

CASE 21, Mrs. K., aged 54. July 21, 1883: R V + 2 = $\frac{4}{9}$; scotoma above and in. L V = hand-movements up and out only; *right,* sclerotomy; *left,* iridectomy. August 24th: *Right,* sharp attack of cloudy vision. March 31st: V R + 2.5 = $\frac{5}{12}$; large scotoma as in Fig. 19.

April 3d: right, iridectomy. June 21, 1886: V R + 2 = $\frac{5}{9}$.

In this case also the patient was not relieved by sclerotomy. For a year after that operation the attacks of glaucoma persisted. The field of vision became more and more restricted, until finally the diseased process was brought to a standstill by an iridectomy.

I have not observed attacks of glaucoma occur after iridectomy, as I have seen them set in after sclerotomy has been performed. Apart from glaucoma malignum, cases of blindness from glaucoma in which an iridectomy had been done, are extremely rare. In my experience, examples of the inefficiency of sclerotomy in this disease are quite common. Observation has convinced me that it is not to be depended upon in cases of true glaucoma. It has been employed most commonly in cases of so-called glaucoma simplex; and it is possible that many a case of stationary or slowly progressive optic-nerve disease with a physiological excavation has been put forward as proof of its curative value. Moreover, the length of time these reported cases of cure had been under observation is much too short. One or two years is not a sufficiently long period when we know that many cases of glaucoma are of much longer duration; the following case, for example:

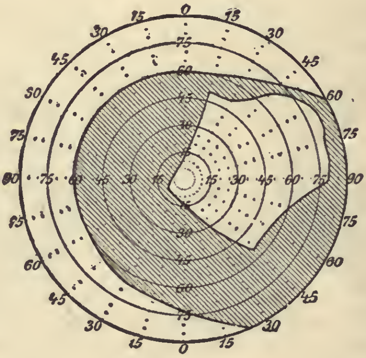

FIG. 19.

CASE 22.—Mrs. J. first presented herself for treatment in 1874. For four or five years previously she had suffered (mostly in the evening) from cloudy vision affecting her left eye. After a night's rest the attack disappeared. The first severe symptoms were noticed February 20, 1874. These were ciliary injection, mydriasis, corneal haze, etc. V = fingers at 1½ *m.* Iridectomy, February 25th. A few days later the right eye was also seized. Patient refused iridectomy; attack soon over. March 24, 1874: L V + 4 = $\frac{10}{30}$ - $\frac{10}{20}$; R V + 5 = $\frac{10}{20}$ - $\frac{10}{15}$. Repeated glaucomatous attacks in *right*, especially a marked seizure in January, 1883. V + 5 = $\frac{1}{5}$. Eserine induced complete myosis. V + 5 = $\frac{5}{5}$. On February 6th another attack; then iridectomy and relief. February 8, 1888: V R and L + 5 = $\frac{6}{18}$ - $\frac{6}{20}$.

In the right eye the so-called "initial stage" had lasted about nine years, from February, 1874, to February, 1883, the date of the iridectomy. Had a sclerotomy been done at the time of the first attack, the operation might have been given the credit of relieving the patient for that length of time.

The following history shows the untrustworthy character of sclerotomy.

CASE 23.—Mrs. W., aged forty-five, came December, 1882, on account of cloudy and rainbow-vision. V R = $\frac{5}{9}$ - $\frac{5}{6}$; V L = $\frac{5}{6}$ - $\frac{5}{5}$. The symptoms were regarded as evidence of glaucomatous attacks, and on December 15th *sclerotomy* was done in both eyes, but the halo-vision and cloudy sight still persisted, until January, 1885, a severe attack of glaucoma affected the left eye, so that V = finger-counting at ½ *m.* After a bilateral iridectomy no more evidence of the disease. April, 1887, V L = $\frac{5}{5}$.

Finally the same disagreeable results may follow both sclerotomy and iridectomy, at least I have seen after sclerotomy retinal hemorrhages so profuse that it would have greatly interfered with any remnant of visual acuteness.

From all this we can only conclude that the essential part of glaucoma operations is the excision of a portion of the iris. It is true that we are ignorant of the real connection between the two, but it is not the province of physical science even to discuss stories that will explain all the phenomena. He who reasons like a *physician* is satisfied therewith. When the array of facts is complete, their interpretation follows as a matter of course.

If the iris is not attached to the cornea, but simply pushed forward as a consequence of increased volume of the vitreous, we may certainly regard the former condition not as a cause, but as a result, of the glaucoma. One may frequently observe in the course of the disease that attacks disappear spontaneously, even while the iris is bulging. The iris resumes its normal position without affecting the further progress of the glaucoma.

Among other untenable propositions which Leber advanced at the International Ophthalmological Congress (Report, 1888, p. 278), the following may be mentioned: "When the filtration angle becomes entirely occluded, no operative procedure is of any value—as is the case in the later stages of glaucoma." This assertion has no foundation in fact, because no man can say whether a complete stoppage of the irido-scleral angle is present during life or not. Experience teaches us not to count upon any good results from an iridectomy if the conducting power of the optic-nerve fibres be destroyed, but that has nothing to do with the filtration angle.

The only definite physiological fact of any value relating to the action of iridectomy in glaucoma that has yet been made is the observation of Exner,[1] that at the border of the coloboma a direct anastomosis between the arteries and veins of the iris is rendered possible, "so that the arterial blood without passing through an intervening capillary area may at once reach the veins." The blood pressure in the iris must in this way be at least moderated; but the claim that this alone suffices to cut short the glaucomatous process is, in my opinion, rather premature.

As soon as an iridectomy has been performed on a genuine case of glaucoma, we may regard it as certain that the disease has been brought to a standstill. The instances in which this is not true are so extremely rare that from a statistical standpoint they do not carry much weight.

Though iridectomy is a safeguard against glaucoma, there are doubtless cases in which marked inflammatory attacks of the disease affect iridectomized eyes. I can recall but one instance in my own experience. I have already reported this case,[1] but will again refer to it at some length.

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[1] *Wiener medic. Jahrbücher*, 1873.

Case 24.—Mrs. A., æt. 66. First saw her July, 1878. Left eye: (H 5.5) V = $\frac{6}{9}$. Right, mature cataract. November, 1882, right: keratitis, œdematous infiltration and iritis, with numerous synechiæ. After four weeks' treatment with atropine the inflammatory symptoms disappeared, leaving several posterior synechiæ—two small ones above, a broad one below. In July, 1885, extraction of the cataract in right eye with a broad iridectomy. The coloboma was perfect, and there was no incarceration of the iris; the inferior synechiæ of course remained. February 8, 1886, the patient returned, on account of pains which she had for several weeks in the left eye. There was a small amount of ciliary injection; some infiltration and haziness of cornea; $+$ 12, V $\frac{1}{12}$ – $\frac{1}{18}$, corresponding to the amount of corneal cloudiness; atropine. The patient lived outside of Berlin, and did not again return until March 6th, with condition but slightly changed. In place of the cornea infiltration there was some loss of substance. At the next examination, March 16th, the area of corneal haze was small, its lower segment showing a superficial loss of substance. Fundus examination, now possible for the first time, revealed marked arterial pulsation, as well as a deep excavation extending to the disk border. Owing to contractions of the orbicularis it was not possible to determine the ocular tension, but after the use of cocaine it was found to be distinctly increased. Left eye: Tn. Hand movements perceived out and above only. In addition to the keratitis superficialis, etc., the patient had glaucoma, which would certainly have been diagnosed earlier had she presented herself with greater frequency. Later, extraction was performed on the left eye. Then discission of a secondary cataract, a prolonged convalescence due to inflammatory complications, and finally a good result.

I lay special stress upon the fact that extraction of the right lens was followed by normal convalescence, and that the eye was not affected by glaucoma until eight months afterwards. Schmidt-Rimpler[2] has observed a similar case. Three months after a perfectly successful peripheral linear extraction (rather large coloboma, no iris incarceration) the eye was attacked by acute glaucoma. There were no initial symptoms. Even in this case the iridectomy was of value. It is worthy of note that as a result of previous inflammatory attacks there were extensive anterior and posterior synechiæ.

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[1] These Archives, vol. xvii., 2, p. 140.

[2] Graefe-Saemisch: "Handbuch der gesammten Augenheilkunde," Bd. v., S. 49.

There are several cases reported in literature in which glaucoma is said to have affected aphakic and iridectomized eyes before the extraction wound had completely healed. I cannot help suspecting that these symptoms, which appeared during convalescence after extraction, were not as closely observed as they might have been. To this category belong many cases which Treacher Collins[1] has well designated "cases which for want of a better name may be termed glaucoma after extraction."

This term may well be applied to certain cases of intraocular inflammation which are conveniently termed glaucoma for want of a better word. But it is not to be wondered at when eyes (whose ailments are styled glaucoma simply because it has an attractive sound) are enucleated to afford opportunities for anatomical research, that they furnish little or no information.

Hydrophthalmus is also a form of glaucoma, and has been recognized as such since Horner demonstrated that it may lead to excavation of the papilla and blindness. During the first stages of hydrophthalmus in early infancy I have noticed the repeated advances and retreats of glaucomatous attacks, the increased tension, the ciliary injection, and the hazy cornea, and recognize in hydrophthalmus a result of glaucoma occurring in infancy and childhood. The increase in the volume of the vitreous stretches the non-resistent sclerotic of infancy. That there is such a thing as a congenital hydrophthalmus is undoubted, and it is common enough to call every case of hydrophthalmus in children congenital. The youngest child that I have ever seen affected by the disease in one eye only was three months old. Whether it was congenital or not cannot of course be decided. This affection must not be confounded with the still rarer *cornea globosa*—

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[1] Ophthal. Hospital Reports, 1888, p. 19.

that is, an abnormally large cornea with a deep anterior chamber, but without stretching and thinning of the sclera.

I have repeatedly performed iridectomy in children affected by hydrophthalmus, and have not found the dangers said to attach to such a procedure at all realized. One should, however, avoid the mistake of making a too peripheral incision, which ought to lie along the corneal border of the limbus. In illustration, I report a few cases.

CASE 25.—R. S., 7 months old, was brought to me March, 1887, with bilateral hydrophthalmus. Iridectomy was finally done on the worst (right) eye the end of December. Four weeks later the left eye presented a dense corneal haze, deep a. c., and sensibly increased tension. For this, an iridectomy was done in January, 1888. Recovery absolutely normal in both eyes. In February, 1889, both corneæ were clear, and visual acuteness, as far as could be determined in a child barely two years old, was good.

It can be proved that hydrophthalmus frequently begins in childhood.

CASE 26.—F. W., æt. 7, came in May, 1889, with hydrophthalmus of the right eye. The disease had been discovered two years before. The sclera about the cornea is stretched, the cornea itself is enlarged and presents a number of opacities; the pupil is wider than its fellow; deep excavation of the nerve; $V = \frac{1}{24} - \frac{1}{18}$. Left, normal; no excavation May 15th; iridectomy *right*, with normal recovery.

As in glaucoma, so with hydrophthalmus, the only remedy that will prevent blindness is an iridectomy made as early as possible. The technique in the latter case is somewhat more difficult than in glaucoma, and it is not easy to draw conclusions as to the final results, since the tender age of the patient precludes a satisfactory test of vision. In the cases operated on by me, convalescence was with few exceptions rapid and normal. Intraocular hemorrhage after iridectomy occurs very rarely.

I will not say more about *glaucoma secundarium* than that it represents too motley a crowd; often the term so used is devoid of any definite meaning. But two groups are distinguishable, the so-called hemorrhagic glaucoma, and glaucoma due to occlusion of the pupil and bulging of the iris periphery.

That glaucoma follows retinal hemorrhage (rarely indeed, though often enough to establish some sort of connection between them) has long been known, but neither the bleeding nor the glaucoma has anything characteristic about it. We know as little why retinal hemorrhage is or is not followed by glaucoma, as why or when (especially if a cloudy cornea prevents an ophthalmoscopic examination) the disease exhibits the hemorrhagic complication. As in primary glaucoma, the hemorrhagic form occurs most frequently in hypermetropia, more rarely in emmetropia, and least often in myopia.

According to reports of many cases, the glaucomatous outbreak occurs a short time—a few days—after the retinal hemorrhages. It may be said in this connection that one cannot tell how long they may have been present. It may easily happen that peripheral bleedings have occurred a long time previously, in which case the patient would not notice them until a hemorrhage in the macular region led to an ophthalmoscopic examination. As a rule, the glaucoma follows the hemorrhage after one to two months, the longest interval, so far as my observation goes, being eight months.

CASE 27.—Mr. S., æt. 72, was treated October 2, 1876, about eight days after he had noticed certain peculiar "glittering spots" before his right eye. Right eye: No. 5.5 is slowly read. Numerous small (diameter $\frac{1}{3}$ of the disk) retinal hemorrhages, some in macular region. Retinal veins tortuous; retina unclouded; vitreous clear; V L (M $\frac{1}{16}$) = $\frac{6}{12}$; fundus normal. May 29th: sight much worse than before, the retina about the optic nerve hazy, and the vessels, especially the arteries, very small. More hemorrhages, some of which appear quite recent. June 5, 1877: Pain and marked pericorneal injection for past eight days; cornea cloudy; iris unchanged; atropine.

June 10th.—Atropine mydriasis; moderate pains; considerable injection of scleral conjunctiva; cornea very hazy; no view of fundus; marked increase of tension. Eserine 1 per cent. June 16th: pupil somewhat irregular but of normal size; otherwise the situation unchanged. Finally, July, 1877, the continuous pain affected the patient's health and the lost eye was enucleated. The result of the anatomical examination is given by Deutschmann.[1]

It goes without saying that the prognosis in hemorrhagic glaucoma is more unfavorable than in retinal hemorrhage. The injury to sight due to the hemorrhage is brought about by a tearing apart of the nervous elements within the substance of the retina, and of course is irreparable; but our experience of hemorrhage setting in after iridectomy teaches us that the retention of good vision is compatible with retinal bleedings. In cases of glaucoma hemorrhagicum the most important question relates to the amount of damage done to vision by the retinal hemorrhage before the glaucoma set in, because the most that can be hoped for is the restoration of the amount that was then present; if there has been useful vision it is our duty to attempt to preserve it. *Iridectomy in glaucoma hemorrhagicum has acquired an undeservedly bad reputation.* Although in some cases under my care a well-timed operation had preserved the visual acuteness, there were others where iridectomy did no good. These latter were instances in which sight had already been lost. I will report some of them.

CASE 28.—Mrs. K., aged 64. September 11, 1873 R H $\frac{1}{20}$; V = $\frac{16}{30}$, + $\frac{1}{10}$ Sn. No. 2 : 12″ L : four weeks before, the house physician noticed that the left eye had become blind from retinal hemorrhage. In addition to this she became the subject of a painful inflammation of the eye, the pains being situated in and over the eye. The cornea is cloudy, the pupil (after atropine) somewhat dilated and fixed by posterior synechiæ. The scleral conjunctiva is injected, but the anterior perforating veins are not dilated. Slight increase of tension. Fingers counted eccentrically in all directions. Fundus not visible.

As the pain increased in violence during the next day, I advised enucleation, but the patient would consent to an iridectomy only. Accordingly, on September 30th, a broad, regular, downward iridectomy was done under chloroform, with the keratome. Shortly after the bandage had been applied and the patient had recovered consciousness, vitreous presented under the dressing.

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[1] *v. Graefe's Archiv*, Bd. xxv., 3, S. 163.

This was probably the result of straining (from vomiting). Severe pains now set in, which were relieved by applications of ice. No doubt an intraocular bleeding had occurred, although this much-dreaded complication is extremely rare. Swelling of lids and conjunctiva followed, with moderate pain. These soon disappeared after treatment with lukewarm fomentations. October 5th: there is no more swelling of the lids; some blood in the anterior chamber. Later on, bulging of the cicatrix, cataract, and complete adhesion of the pupillary border; the a. c. remained empty. The pains are slight; some persistent increase of tension.

In July, 1874, these symptoms were for a time more urgent, the inflammatory condition more marked, and one spot, in the upper and inner ciliary region, painful on pressure. Finally atrophy set in with flattening of the globe in the direction of the recti muscles. No more complaints.

The condition of the right eye entirely unchanged.

Last examination, March 24, 1879: Left, phthisis bulbi; ciliary soreness above, occasionally also spontaneous pains.

R + $\frac{1}{12}$ No. 0.5 – 0.75. Papilla not excavated; F of V uncontracted; commencing nuclear cataract.

In another and more disastrous case of this kind, where the author did an iridectomy in one eye and Hirschberg operated on the other, malignant glaucoma set in and complete loss of sight was the result. In this instance there was a complication of retinal hemorrhage with malignant glaucoma, from which one may conclude that one affection does not necessarily exclude the other.

I have notes of six cases in which the iridectomy wound healed readily and without complication, but either more acuteness of vision or less resulted than might have been expected. One of them I give.

Case 29.—Mrs. M., aged 47, came March, 1887. V L (H 1.5) = $\frac{6}{12}$. Oph. normal. R: for three weeks vision getting worse; numerous retinal hemorrhages. V = fingers at 15 *cm.* Middle of May: R: pains in and redness of the eye. 26th: smoky haze of cornea; pupil dilated and fixed; marked increase of tension; fingers counted at 10 *cm*; hand movements perceived eccentrically everywhere.

June 19th.—Iridectomy followed by normal recovery.

In all these six cases the purpose of the iridectomy was completely accomplished, *i. e.*, the pains were relieved and whatever remained of sight was preserved. When useful vision is still present in a case of glaucoma hemorrhagicum, it can be maintained by means of iridectomy.

On account of the presence of a pressure-cavity it is legitimate to speak of secondary glaucoma as following iritis. The conditions present are complete occlusion of the pupil and bulging of the iris periphery.

Glaucoma and iritis are, of course, not mutually exclusive, and they may develop together, as in cases that have come under my observation.

I know of others, also, that had been treated by atropine on account of synechiæ until they became blind from glaucoma. The corneal cloudiness in such cases is quite the same as that occurring in glaucoma; the atropine mydriasis excites no comment, the fundus is not visible, and the examination of the tension is really the only means available for a differential diagnosis.

Quite frequently, on the other hand, one sees loss of vision occur in cases of pressure-excavation where complete occlusion of the pupil and pushing forward of the periphery of the iris had developed. It is when iritis is followed by these additional symptoms that glaucoma is especially to be dreaded. Its cause lies in the dragging upon the ciliary body. If, however, this state of affairs comes about slowly and without noticeable inflammatory signs, there is, in the early stage at least, no danger of glaucoma, especially when a prophylactic iridectomy is made as soon as possible. To show how pupillary occlusion with prominence of the iris periphery can develop without leading to glaucoma, I report the following case:

Case 30.—Mr. G., about 30 years old, consulted me, middle of May, 1882, about a right-sided iritis which had already lasted three weeks. The patient was treated with atropine locally, and, on account of fibrinous exudation in the a. c., was ordered Turkish baths with good results. About June 15th a return of the iritis. The end of July the disease had disappeared; V — 0.75 = $\frac{6}{8}$. June, 1887, another iritic attack with a small posterior synechia in the upper part of the pupil. End of July, 1890, still another attack of iritis in the right eye. This time there was marked conjunctival hyperæmia with some corneal haze and pain. Atropine and duboisine produced complete mydriasis except in the region of the synechia above mentioned; $V - 0.75 = \frac{5}{25}$; increased cloudiness of cornea; lessened mydriasis. Finally, after a severe and prolonged attack, although the mydriatics were discontinued, the pupil remained dilated. The V gradually recovered and was from $\frac{1}{18}$ to $\frac{5}{15} - \frac{5}{10}$ the middle of October. Towards the end of November, on account of some accommodative paresis, eserine was instilled, but was stopped in December, having meantime produced a marked but temporary contraction of the pupil. Jan. 10, 1891, $V\,R - 1.5 = \frac{5}{15} - \frac{5}{10}$. Pupil in same condition as before, but is funnel-shaped, its entire border being bound down to the anterior capsule. Its periphery is everywhere bulging, but there is no conjunctival hyperæmia, no increased tension, no pain, etc. January 14th, iridectomy. January 28th, $V\,R - 1 = \frac{5}{15}$. This iris is now in its proper plane, and on careful examination the site of the pupil is seen to be occupied by a delicate membrane dotted over with fine dark points. The artificial pupil is black and clear.

The bulging of the iris periphery had in this case lasted a full week without the faintest sign of glaucoma.

The claim that blocking of the filtration angle in the anterior chamber is the cause of glaucoma is put forward with all the authority of a physiological law. But natural laws permit of no exceptions, and it must necessarily follow, if Leber's theory were correct, that every case of a narrowed irido-scleral angle is followed by glaucoma. But this is by no means true. Every anterior synechia, inasmuch as it brings about a lessened filtration angle, would result in glaucoma, yet the fact is that these two conditions are almost never associated.

Priestley Smith[1] puts forward in support of the filtration-angle theory the following clinical evidence: "A soft, transparent lens, incompletely attached at the zonula, slips into the anterior chamber and lies in front of the pupil. Inside of an hour a severe glaucomatous seizure. Next day the lens returns to its place and the glaucoma disappears. This is repeated upon several occasions and is followed as often by a return and a subsidence of the glaucoma. Finally the lens is extracted and the glaucoma does not return." Dislocation of the lens into the anterior chamber is a well-known cause of severe and painful inflammatory symptoms which I would prefer to regard as an incipient irido-choroiditis.

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[1] International Congress at Heidelberg, 1888, p. 231.

Glaucoma presents an entirely different picture. That an anteriorly dislocated lens is capable of completely obliterating the filtration angle is at least a rather strong statement. In my opinion the backward pressure of the lens *against* the iris *from* the cornea would rather tend to enlarge it. Moreover, the lenticular diameters are less than those of the anterior chamber. If the body of the lens, only partially occupying the anterior space, produces glaucoma, why does not every anterior synechia bring about a similar result? Fig. 20 (*l. c.*, p. 232) represents the diagram with which Priestley Smith illustrated his case, but, as Samelsohn (*l. c.*, p. 280) observed, during the discussion that followed, it is not a faithful picture of recent cases of the kind. Adhesion of the iris to the cornea, as shown in the picture, may occur later, but it is a consequence of the inflammation and not a cause of it.

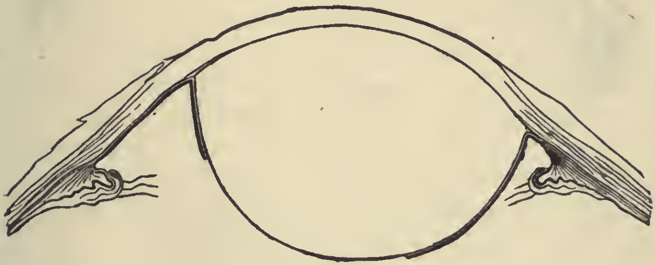

FIG. 20.

Dislocation of the lens *into the vitreous* is said to produce secondary glaucoma. I consider many of these cases to be traumatic in character, while others are members of that group that has been labelled glaucoma "for want of a better name."

For the purpose of making the filtration-angle theory do service in the case of vitreous dislocations of the lens Priestley Smith (*l. c.*, p. 282) pictures the condition represented by Fig. 21. The filtration angle is said to be obliterated on one side by pressure of the lens on the ciliary body, on the other side by the displaced vitreous. One may well ask, what makes the lens press on the ciliary body? The weight of the displaced lens is all that can be exerted upon it, and that the ciliary region can stand. If the normal pressure of the vitreous body leads to glaucoma, every man and every other animal would have the disease.

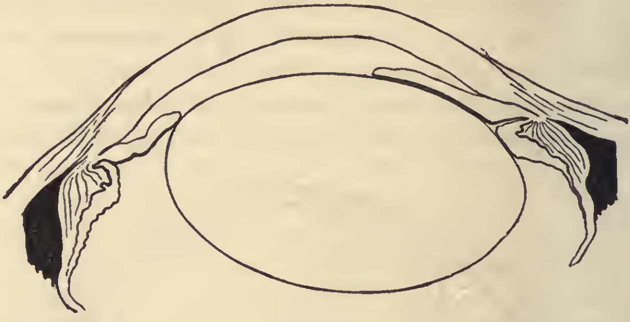

FIG. 21.

That lenticular luxation may exist for years without evil consequences is shown by the following case:

Case 31.—Mr. L., æt. 44. September, 1874, V R = $\frac{10}{30}-\frac{10}{40}$; V L = 1. In May, 1875, the right lens was cloudy throughout. In June, 1875, it was noticed to be movable and dislocated downwards. Beginning of August, with + 10 D V = $\frac{1}{30}-\frac{1}{40}$. In October it was completely displaced downwards and visible as a light-gray body. The vitreous is normal; only a slight circumscribed haze near the papilla. July, 1880, V R + 10 = $\frac{5}{12}-\frac{5}{9}$; left, irregular striæ in anterior cortex; V = $\frac{5}{9}$. January, 1883, V R + 10 = $\frac{5}{9}-\frac{5}{6}$; left, mature cataract. The left lens gradually sank into the vitreous. January, 1890, V L + 9 = $\frac{5}{18}$.

This patient, according to Priestley Smith, should have had glaucoma, but he did not. The condition just referred to, long recognized and first described by Kniess,[1] is no proof of the assertion that it is a cause of glaucoma; there is abundant evidence to prove that it may exist without glaucomatous symptoms. Clinical evidence, for example: the anterior chamber is sometimes so shallow in interstitial keratitis that anterior synechiæ may form and the filtration angle be obliterated; yet no glaucomatous signs develop. On the contrary, the tension is usually minus in such cases. Likewise, Leber's theory, that in eyes enucleated because of glaucoma the drainage from the anterior chamber is considerably lessened or entirely choked proves nothing, except that an enucleated glaucomatous eye remains harder a longer time than one not affected by glaucoma, because the filtration angle is obliterated by the glaucoma, in consequence of the increased volume of the vitreous.

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[1] *v. Graefe's Archiv*, Bd. xxii., 3, p. 163.

The experiments by Leber[1] on the filtration of fluids at the irido-scleral angle are post-mortem experiments on animals, and of little value when living eyes are under consid-ation. His experiments on the latter simply show that a *slight* increase of intraocular pressure may disappear, but marked tension not. It does not follow from this that gradual decrease of tension with a moderate increase of pressure comes about in consequence of filtration through the filtration angle, for if the manometric pressure in the anterior chamber is increased absorption takes place, as Donders'[2] experiments show, as a result of the communication of the pressure to the lens and vitreous. At any rate this absorption process has nothing to do with the filtration angle, as pressure on the sclera and vitreous humor pushes forward the lens and closes the angle.

The most important consideration in the investigation of glaucoma is that of a correct diagnosis. If that is right no ornamental addition is called for.

It is senseless to call one and the same case glaucoma simplex to-day and simple glaucoma "with inflammation" to-morrow, as well as to talk of the possibility of every acute glaucoma becoming chronic, and every chronic glaucoma becoming acute. Doubtless there are differences between primary and secondary glaucoma, but one should use the latter term with great care, lest he designate totally different conditions by the same term, as has often happened in glaucoma simplex, and not infrequently occurs in inflammatory states.

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[1] *v. Graefe's Archiv*, Bd. xix., 2, p. 87.

[2] *v. Graefe's Archiv*, Bd. i., 2, p. 101.

TRAUMATIC RUPTURE OF THE SCLERA IN THE ANTERIOR HALF OF THE BULBUS.

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(*With three wood-cuts and eight figures on plates xi.–xiii. of vol. xx., June edition.*)

Translated by Dr. J. M. MILLS, New York.

THE attention which opthalmologists have bestowed, for a long time, upon the rupture of the foremost zone of the corium, has its motive especially therein that from these injuries changes are often produced similar to those caused by our cataract extraction. I gathered together from the literature[1] at least 144 cases, and, in addition, six from my own observation.

Among 97 cases of which I obtained accounts of the instrument of assault, 23 were caused by a blow from the horn of a cow or ox (see accompanying wood-cuts ; Figs. 1 and 2, ends of cow horns ; Fig. 3, of an ox horn), 19 by a blow or fall against the edge of some irregular object, 14 by a thrust or blow with the end of a wooden or iron bar or other similar instrument, 8 by a blow with the fist, 6 by a thrust or pressure of a finger against the eye, 1 by a push against the outstretched hand, 9 by pieces of wood flying into the eye, 3 by stones or earth-clods thrown into the eye, 4 by a blow from a horse's hoof ; 3 were struck with a whip ; 1 by a knock against the door-knob ; 1 each by a blow with a hammer, knuckle-duster, rapier, and wand.

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[1] In the older literature (beginning and first half of the present century), are found intimations of cases belonging to this class, though their complication with aphakia was not recognized, etc., etc.

The uniformity, just mentioned, in most of the cases, renders superfluous the presentation of more detailed histories.

Notwithstanding the large number of individual observations, the explanation of the occurrence of the indirect rupture of the sclera is still a matter of dispute. Whereas until a short time ago the opinions of ophthalmologists were divided between the theories of Manz,[1] Schröter,[2] and v. Arlt's[3] theory of flattening, now the latest investigator in this region, Hughes, endeavors to give this problem a purely mechanical solution.

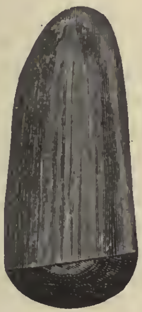
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FIG. 1.

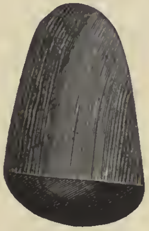
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FIG. 2.

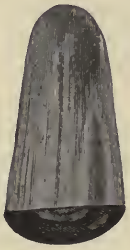
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FIG. 3.

Twelve years ago (at the time of the appearance of the large hand-book of Gräfe-Sämisch) the pathologico-anatomical knowledge concerning our injuries was exceedingly meagre; and although since then it has been greatly enriched by the excellent contributions of Alt,[4] Treitel,[5] and Schäfer,[6] they must still be considered deficient, as the examinations of the authors just mentioned consisted only of eyes that had to be enucleated on account of intense inflammation or threatening sympathetic affection, whereas the anatomical condition of an eye that was cured (with retention of sight) has, until now, been wanting.

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[1] Manz: "Zwei Fälle von Traumatischer Bulbusruptur."—*Klin. Monatsbl.*, Bd. iii. (1865), S. 170.

[2] Schröter: "Zur Entstehung der Traumatischen Scleralruptur."—*Klin. Monatsbl.*, Bd. iv. (1866), S. 242.

[3] v. Arlt: "Ueber die Verletzungen des Auges," etc., Wien, 1875, S. 5–12.

[4] Alt: "Beiträge zur Patholog. Anatomie, Fall 3."—*Gräfe's Arch.*, xxxiii., 3, p. 26.

[5] Treitel: "Beiträge zur Patholog. Anatomie des Auges."—*Arch. f. Ophth.*, Bd. xxvi., 3, S. 109.

[6] Schäfer: "Aniridia et Aphiatraumatica."—*Arch. f. Ophth.*, Bd. xxix., 1, S. 13.

I hope to supply this want by the following somewhat more detailed description of the first case, and if I add thereto pathologico-anatomical communications of other eyes, injured in the same manner, but affected with severe inflammation, it is only because the contrasting of the different terminations of the same injuries would not be without profit, and also the number of autopsies appertaining thereto is still very small.

CASE I.—The eye was removed on December 12, 1883, by my colleague, Dr. Purtscher, in Klagenfurt, from the body of a more than 60-year-old woman. Fifteen years before, she was injured in the right eye by a thrust from the horn of a cow. Dr. Purtscher, who kindly presented me with this specimen, noticed, on the body, the typical iris coloboma, upwards and inwards, but was not in a position to obtain further clinical details. The left eye of the subject was normal and was not enucleated.

The normally formed bulbus, after remaining several years in Müller's fluid, was further hardened by me in alcohol, then opened by an equatorial section, upon which the fluid part of the vitreous humor escaped. Upon examination of the anterior half of the bulbus, from within (Fig. 1), the absence of the lens is right away noticeable, instead of which a transparent diaphragm, consisting of the more consistent parts of the vitreous, covers the posterior surface of the iris. There is a defect in this, directed upwards and somewhat inwards.

The anterior half of the bulbus was imbedded in celluloidin for the purpose of microscopic examination, and a series of sections made parallel to the plane of the middle line of the coloboma.

An arched cicatrix occupies the place of the scleral rupture, which encircles the upper half of the corneal periphery.

The rupture lies, in its whole extent, in the scleral border of the cornea, beginning in the medial line, almost on the horizontal meridian, ending laterally somewhat above the same, and penetrates at its lateral end only the superficial scleral layers, whereas otherwise it perforates every part. Schlemm's canal is obliterated. The cicatrix attains its maximum width somewhat medial from the vertical meridian. The cicatrix consists of non-vascular fasciculi of connective tissue that have mostly a meridional direction, are not particularly rich in nuclei, are partly straight and partly slightly undulating; the cornea is unchanged. The sclera adjacent to the cicatrix is somewhat thinned and more strongly arched. The conjunctiva is adherent to the cicatrix, but is not ruptured. The scleral cicatrix inclines obliquely, from behind—upward towards the front—and downward, its proximal edge deeper than the distal, and covered by the ciliary processes (Fig. 2, Plate xiii.). Not only the apices of the ciliary processes, but also the vitreous body, are connected with the scar. Somewhat posterior to the place of rupture there is in the scleral tissue, thickened by cicatricial bands, a cavity which contains the lens capsule, denuded of its epithelium and very much wrinkled. Back of the lens cavity, directly episcleral, a strip of pigmented tissue has healed into the wound, and can be recognized as a remnant of iris by its vessels and adherent uveal pigment. The corpus ciliare of the opposite side is normal. On the medial, as well as on the lateral side, the ciliary process and vitreous are more extremely incarcerated in the cicatrix. On the nasal side the iris, doubled up, is wedged in the scar (Figs. 3 and 4); at the periphery it is ruptured (iridodialysis). Near the lateral periphery of the chamber the rupture penetrates the superficial fasciculi of the sclera. Schlemm's canal is intact, whereas, on the contrary, the sinus of the chamber is altered in such manner that the processes detached from the root of the iris are drawn near to the inner wall of Schlemm's canal, but are not adherent. Thereby also the anterior triangular side of the corpus ciliare appears shortened. The foremost portion of the retina shows cystoid degeneration, whereas the posterior section, as well as the optic nerve, are entirely normal.

CASE 2.—M. Georg. Entered v. Jäger's clinic on Oct. 24, 1875. Twenty-three weeks previously the left eye was injured by a piece of wood flying against it. Six weeks after the injury suffered intense and continuous pain. Thereupon began an inflammation in the right eye that, at the time of admission, was recognized to be a pronounced sympathetic iridocyclitis. The left injured eye was atrophied, soft, and without a lens; above the corneal margin was a swelling that contained the dislocated lens. Enucleation on October 6, 1875.

The anterior half of the bulbus, left to me, was cut in half by a vertical meridional section. The sclera in the region of the rupture is thinned, otherwise thicker than normal. The rupture, almost in its entire extent, passes through corneo-scleral margin, so that the edge of the scleral wound corresponds about to the insertion of the ciliary muscle, and Schlemm's canal is included in the rupture. Only at one end the rupture lies farther backward, and only the sclera is involved, the uvea remaining intact.

The cicatricial mass consists of tough, nucleose reticular connective tissue, which unites the lips of the wound and connects externally with a pouch, the empty bed of the dislocated crystalline body (Fig. 5, *cfl*), and internally with a pseudo-membrane lining the ciliary body (*cye*, *cye'*). The iris is partially imprisoned in the scar, partially detached and tilted back.

The ciliary body is infiltrated in every part by a very extensive inflammatory product. Below, the extremely swollen processes of the posterior surface of the iris come so close together that they almost touch, whereas, on the side of the rupture, they show a much less degree of turgescence. The fascicula of the ciliary muscle are pressed apart; the form of the triangle of the muscle is changed at the apex by the enlargement of the angle; the nuclei on and between the fasciculi of the muscle are considerably increased. The tissue between the muscular and pigment layer exhibits an almost continuous deposit of round cells, in which hardly anything of the matrix can be distinguished. The layer of cylinder cells on the pars nonplicata corp. cil. is partly detached from the pigment-layer; the individual cells are considerably elongated, and have grown out into fibre-like formations. On the inner surface of the corpus ciliare and on the posterior surface of the iris are lodged masses of exudation that contain in the delicately fibred matrix numerous round and spindle-cells. The cyclitic membrane sends off shoots toward the axis of the eye.

The infiltration of the corpus ciliaris extends to the foremost part of the choroid, which is considerably thickened and appears to be permeated by round cells, that in places are massed into regular pus-nodules. Partly behind the iris and partly near the margin of the pupil, projecting into the anterior-chamber space, are extensive fibrinous coaguli, which contain numerous leucocytes in the marginal portion, but only a few in the middle. A similar very large coagulum lies eccentric, near to the corpus ciliare of the ruptured side.

There is not even a trace of the zonula Zinnii visible. The anterior section of the retina has become adherent to the previously mentioned layer of exudation; it is detached and very much wrinkled, its structure materially altered by hypertrophy of the basilar fibres, and by numerous larger and smaller hemorrhages that have undergone degeneration.

Case 3.—This eye was enucleated at v. Jäger's clinic in Vienna. Unfortunately there is no account of the nature of the injury or of the clinical condition.

The bulbus was cut in half by horizontal meridional section, and has a sagittal diameter of 24 *mm*, and a horizontal of 21 *mm*, the other a sagittal diameter of 24 *mm*, and a horizontal of 23 *mm*.

Medially on the corneal border is a rupture of the sclera plugged by a cicatricial mass, the edges of the wound being very much curved in (Fig. 6). A capsule of connective tissue is connected externally with the cavity of the dislocated lens (*cfl*). The iris is applied to the posterior surface of the cornea. The rupture is situated exactly on the corneo-scleral margin, comprising almost half of the conical periphery. Internally the rupture is plugged by new-formed connective tissue connected with the ciliary body, and the adjacent part of the detached retina, constituting a very much wrinkled pseudo-membrane that is not sharply separated from the cicatrix; it is permeated by numerous hemorrhages from new-formed vessels, and contains accumulations of pigment together with gland tube-like proliferation products of the cylinder-epithelial layer. Pupil obliterated. The remnant of anterior chamber is empty.

The corpus ciliare is infiltrated with leucocytes. The cord of the optic nerve is thinner, the nerve-fibre bundle narrower and more granular. The connective-tissue septi are thickened and very rich in nuclei. The intermarginal space (particularly distinct at the bulbar extremity) is enlarged; the arachnoidal sheath, thickened.

CASE 4.—The right eye of L—— G——, on January 10, 1885, injured by the horn of a cow. Immediate blindness. Enucleation twenty-four days later. Globe atrophic. In the inner-upper scleral border is a rupture 10 *mm* long, 2–4 *mm* wide, curving 3–4 *mm* backward. Cornea flat. Ciliary body and iris absent at the place of the rupture.

Microscopic.—The lumen of Schlemm's canal, on the side of the rupture, is destroyed. The retina is in the wound. Cyclitic membrane. .Corpus ciliare more atrophied than inflamed. The adjacent choroid thickened by contraction and dense leucocytic infiltration. The optic nerve presents, at its intraocular extremity, evidence of inflammation; in its orbital portion, the picture of atrophy.

CASE 5.—P. B. sixty-six years old, l. e. hurt with the finger, intense pain, total blindness. Rupture of the sclera near the medial part of the cornea. Eye removed thirty-three days later. (See Fig. 7, Plate xii. and Fig. 8, Plate xiii.)

Around the rupture there is considerable hemorrhage (*sg.* Fig. 8.) in the adjacent structures. Wound is not cicatrized. Schlemm's canal obliterated. Retina and choroid detached. Lens absent.

CASE 6.—Z. M., forty-eight years old, on May 21, 1881, l. e. struck and became blind immediately. Great pain for four weeks. In seven weeks right eye inflamed. Iritis (sympathetic?). Medially and 1 *mm* behind the cornea, a subconjunctivo-scleral rupture 8 *mm* long, 3 *mm* wide. Eye enucleated two months after the injury.

Lens absent; also the iris near the wound. Choroid detached. Ciliary body protruding through the scleral wound. Choroid infiltrated with pus cells. Only the first eye recovered and probably preserved good sight permanently. In the subconjunctival rupture the iris and ciliary processes were incarcerated. In cases 2 and 3 severe irido-cyclitis occurred despite the subconjunctival rupture of the sclerotic. Yet small lacerations of the conjunctiva, not discoverable by the anatomical examination, may have existed.

If the scleral wound is made an open one by a simultaneous rupture of the conjunctiva, then are the chief conditions for an infection present, but when occasionally such cases have a favorable course, then is it due either to happy chance or to the asepsis used which kept the phlogogenic agencies from the eye.

It is much more difficult to understand those cases in which an eye, with rupture of the sclera having induced a sympathetic affection of the severest form, itself, however, remains entirely or almost free from inflammation. Such observations are communicated by Schrag, Jacob, Bresgen, Arlt, and Manolescu.[1]

Also these authors will accommodate themselves to the microphytic theory, but it remains for the future to prove if the explanation, given by Deutschmann,[2] the exact and circumspect originator of the migration theory, is more than a mere digression from the facts. "On the other hand it is just in this acceptance of this infection theory, not excluded, that in rare instances the cells of the organ wage a successful war with the micro-organisms, that have entered, for the preservation of the eye. So that the first affected eye, though unable to prevent the rapid propagation of the germs along the optic nerve apparatus, may nevertheless finally render the germs it contains harmless, and thereby preserve a high degree of valuable functional power; whilst the eye secondarily affected, succumbs to the micro-organisms with which it is infiltrated."

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[1] Schrag ("Einige Fälle von Rupturen der Sclera und Choroidea," J. D., Leipzig, 1870, Fall 3). Right eye: Partly open, partly sub-conjunctival situated scleral rupture. Lens luxated into the vitreous. S. with $+ \frac{1}{2}$ Jg. 5. Left eye: Sympathetic irido-cyclitis, S. Jg. 6.

Jacob ("Some Varieties of Luxation of the Crystallin." *Brit. Med. Jour.*, March, 1870, S., 226). Sub-conjunctival luxation of the lens upward. S. retained. The other eye has been lost by sympathetic inflammation.

Bresgen ("Zwei Fälle von sympathisch. Erkrankungen." *Wiener med. Wochenschr.*, 1878, S. 1197). Right sub-conjunctival scleral rupture. Lens luxated into the vitreous. S. $\frac{20}{50}$. Left sympathetic iritis with opacities of the vitreous, healed without enucleation of the injured eye and with retention of the normal S.

Manolescu ("Aniridie et aphakie traumatiques." *Arch. d' Opht.*, 1885, Bd. v., No. 3, Mai–Juni, S. 227). S in injured, = fingers in 9 meters. In the other eye sympathetic irido-choroiditis, that has run its course. S, after repeated operations, fingers in 50 *cm*.

Besides these there are among the 120 cases still 5 in which the injury had caused sympathetic ophthalmia, of these there were 4 in which the scleral rupture was sub-conjunctival.

[2] Deutschmann: "Ophthalmia Migratoria," Hamburg, 1889, S. 133.

The deviations from the typical positions and direction (circumcorneal) of the rupture are as remarkable as they are rare; for instance, v. Stellwag[1] and Fano[2] describe a rupture that, following the direction of a meridian and laterally situated in the anterior scleral zone, nevertheless permitted the lens to pass under the conjunctiva, and in one case of Schröter's,[3] joined on to the posterior end of an indirectly produced wound (having also a meridional direction), was a second angular rupture—deviating from the former and forming with it a flap.

That the scleral rupture in the large majority of all the cases belongs to the upper periphery of the bulbus has been a well-known fact for a long time.

From the grouping of the material published we ascertain that the situation of the rupture was above 36 times, above and inwards 21 times, above and outwards 11 times, inwards 20 times, inwards and downwards twice, outward 8 times, and outwards and downwards once.

The length of the rupture was uniformly so extensive that it spanned nearly $\frac{1}{3}-\frac{1}{2}$ of the corneal periphery; clinical experience also teaches that the rupture exceeds in length, on the average, a cataract section situated in the limbus.

The situation of the rupture, as described, necessitates almost constantly the participation of the iris in the injury;

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[1] v. Stellwag: *Ophthalmologie* 1856, Bd. ii., 1, S. 468, Anm. 218.

[2] Fano: "Lussazione sotto congiuntivale del cristallino," *Annal. di ottalm.*, Bd. ix., 3 u. 4, S. 364.

[3] Schröter, *l. c.*, Fall 4.

this is almost constant, as in the published statistics there are only two cases in which the lens had escaped through the rent of the foremost scleral zone without destroying the circular form of the pupil.

The contrast to the intact-remaining iris is offered by the complete aniridia.[1] Among 83 cases it is mentioned 17 times, with exact information of the condition of the iris; frequently there remained besides very insignificant remnants of the iris. In only two reports is the dislocation of the completely detached iris under the conjunctiva mentioned (Haenel[2] and Homburg[3]); more frequently the iris escapes through the conjunctival wound at the same time with the lens.

That a true coloboma of the iris occurs is proven by the first communicated autopsy; the shanks of the coloboma, from which the border of the sphincter is absent, are recognized as the edges of the rupture by their covering with hyaline tissue, that is foreign to the normal pupillary border. The section torn from the continuity of the iris-breadth was at the same time detached from its ciliary insertion and partly healed in the pocket-like protrusion of the cicatrix and was considerably shrunken.

In an isolated observation of A. v. Gräfe,[4] a relatively small vertical piece, broken from the equatorial region of the lens, escaped through a scleral rupture under the conjunctiva, whilst the principal mass of the lens system remained intraocular and in its normal position.

I am much less inclined to harbor the illusion that my few suggestions have made entirely clear the mechanism of the rupture of the sclera, as I myself still foster the conviction that the final decision rests with the experiments employed.

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[1] (a) Dixon, James, *The Lancet*, Nov. 27, 1852, cit. n. Massie, Étude, etc. (b) Bericht der Arlt'schen Klinik von Tetzer, Becker und Rydel, 1866, *Wiener med. Presse*, 1866, S. 1205, Fall 2.

[2] Haenel, Jahresber. der Gesellsch. f. Natur- u. Heilkunde in Dresden, 1881 S. 30.

[3] Homburg, "Statistik und Casuistik der Augenverletzungen" (aus Hirschberg's Material), *Inaug.-Dissert.*, Berlin, 1883, Fall 376.

[4] A. v. Gräfe, "Beobachtung einer partiellen Dislocation der Linse unter die Conjunctiva," *Arch. f. Ophth.*, Bd. iii., 3, S. 369.

Explanation of the Figures 1–9.

Fig. 1 (Case 1). Inner view of the anterior half of the bulbus. The vitreous remnants are not drawn. Twice magnified. *c*, cornea; *ci*, cicatrix; *i*, iris; *mco*, *mco'*, medial and lateral coloboma margin; *os*, ora serrata; *pc*, processus ciliaris; *s*, sclera.

Fig. 2 (Case 1). Section parallel to the middle line and somewhat lateral to the medial border of the coloboma. Verrick, obj. 2, ocul. 1. *c*, cornea; *ci*, cicatrix; *cj*, conjunctiva; *cl*, capsule of lens; *cv*, vitreous; *i*, episcleral dislocated piece of iris—fragment of iris in the conjunctiva; *mc*, musculus ciliaris; *pc*, processus ciliaris.

Figs. 3 and Fig. 4 (Case 1). Section somewhat medial from the middle line of the coloboma and parallel. Fig. 3 magnified. Fig. 4, Verrick, obj. 2, ocul. 1. *c*, cornea; *ci*, cicatrix; *cj* and *cj'*, conjunctiva; *cc* and *cc'*, corpus ciliare; *cv*, vitreous; *i* and *i'*, iris; *md*, Descemet's membrane; *pc*, processus ciliaris.

Fig. 5 (Case 2). Vertical meridional section magnified. *c*, cornea; *cc* and *cc'*, corpus ciliare; *ci*, cicatrix; *cj* and *cj'*, conjunctiva; *cye* and *cye'*, cyclitic exudation; *cfl*, fibrous capsule of the dislocated lens; *fc*, fibrinous coagulum; *i* and *i'*, iris; *md* Descemet's membrane; *r*, retina; *s* and *s'*, sclera.

Fig. 6 (Case 3). Horizontal meridional sections. Highly magnified. *c*, cornea; *cc* and *cc'*, corpus ciliare; *ci*, cicatrix; *cj* and *cj'*, conjunctiva; *cfl*, fibrous capsule of the dislocated lens; *cl*, structureless capsule of the lens; *ecy*, cyclitic pseudo-membrane; *i* and *i'*, iris; *r*, retina; *s* and *s'*, sclera; *sa*, anterior synechia; *spch*, suprachoroid.

Fig. 7 (Case 5). Inner surface—view of the upper end of the rupture—magnified twice. *c*, cornea; *ci*, conjunctiva; *md*, edge of Descemet's membrane; *rs*, scleral rupture; *s*, sclera; *ss* scleral groove; *sw*, scleral eminence.

Fig. 8 (Case 5). Section through the middle of the rupture and perpendicular to the course. Fifteen times magnified. *cj*, conjunctiva; *lcj*, limbus of the conjunctiva; *rcj*, conjunctival rupture; *eps*, episcleral tissue; *i*, iris; *mc*, musculus ciliaris; *pc*, processus ciliaris; *s*, sclera; *sg*, blood.

ON PAPILLOMA OF THE CONJUNCTIVA.

By Dr. SIGMUND FUCHS.

FROM THE EYE-CLINIC OF PROF. ERNST FUCHS IN VIENNA.

Translated by Dr. Ward A. Holden, New York.

(*With plate xiv. of vol. xx., German edition.*)

THE tumors which I shall describe here belong to the general group of proliferation tumors or growths (Virchow), and to that special class which genetically at least are composed chiefly of connective tissue.[1]

Commonly called papilloma (Krämer), the more fitting name is fibroma papillare (Virchow). In many cases the epidermoid or epithelial strata make up no small component of the tumor, particularly in those papillary fibromata which develop as a plate or skin-like mass of connective tissue.

In some of these cases the connective tissue forms so small a constituent of the tumor and the epithelium so predominates that the fibrous character of the growth might be disputed, but a study of the genesis of the tumor always shows that in reality it belongs to the class of fibrous growths, and the small amount of fibrous tissue present is due to the fact that there has been a retrograde metamorphosis or an atrophy caused by the excessive development of the epithelial constituent.

Papilloma may develop in any portion of the conjunctiva, and since all portions of the conjunctiva do not possess papillæ, it cannot always be regarded as a simple hypertrophy or as an excessive development of normal papillæ. In the initial stage all these papillary growths appear as a small round knob due to excessive development of the superficial tissue; vessels push into the connective-tissue centre of the little bud and at the same time the epithelial layer grows thicker. New sprouts appear on the bud, and become vascular, forming finally a branched papilla. The papillomata which spring from the tarsal conjunctiva often assume a cauliflower appearance with a very wide base, since the process just described takes place at a number of different points. Similar forms of less extent are seen on the plica semilunaris. Those developing on the bulbar conjunctiva and in the fornix are, on the contrary, branched pediculated vegetations with a papillary surface, the whole having rather a polypoid form.

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[1] Virchow, "Geschwülste," vol. i., p. 287 *et seq.*

What has just been said will explain the similarity of papillomas of the conjunctiva to the pointed condyloma, and will show that it is akin to the warty formations of the dermatologists.

Fibroma papillare of the sclero-corneal border in its initial stage can hardly be distinguished clinically from epithelioma, but when it has reached a certain development and overlaps the cornea, the involvement or non-involvement of the substance of the cornea is a diagnostic sign of great value. For a papilloma which overlaps the cornea may be lifted up and pushed back to the conjunctiva, *but an epithelioma will infiltrate the corneal substance and rest immovably on it.*

Another valuable point is the enlargement of the neighboring lymphatic glands, which, of course, never occurs with papilloma. In all doubtful cases histological examination is required for an accurate diagnosis.

The papillomata of the limbus are distinguished from so-called spring catarrh by their softness and their papillary surface. The individual papillæ have always a small base and a broader extremity and are somewhat movable. Spring catarrh is characterized by photophobia, by its regular recurrence in the spring, and by the fact that it only affects children and adolescents.

No certain causes for the development of papilloma are known; I would refer the reader to the paper by Elschnig[1] for information on this point.

The tumors are benign, although inclined to recur if not radically removed. In no case observed did the growth become sarcomatous or carcinomatous, and the microscopic examinations never revealed evidences of any such change.

The only treatment is the operative removal of the growth, together with the underlying conjunctiva, to guard against recurrence.

The literature on hard and soft fibroma has been collected by Elschnig.[2] An exquisite case of papilloma of the conjunctiva was reported by Magnus in *Zehender's klin. Monatsbl.*, vol. xxv., 1887, p. 384.

The material for these investigations was composed of eleven cases of papilloma seen in Prof. E. Fuchs' clinic in the course of the years 1887–1888; two of these cases were observed for a considerable time, the nine others occurred in out-patients. Details regarding age of patient, situation of growth, duration, etc., were not kept in the latter cases. Histologically they were all similar.

CLINICAL REPORTS.

Case 1.—M. Hansl, æt. 28, presented himself in July, 1887, with a small tumor at the left internal canthus which was removed. In August he noticed a return of the growth and in October the condition was as follows: The lower lid is slightly everted and rising above its margin is a finely papillary red neoplasm; a similar growth is seen at the middle of the free margin of the upper lid. The tumor spread over the tarsal conjunctiva of the inner half of the lower lid as a narrow stripe, and at the inner canthus extended over the fornix to the bulbar conjunctiva. The upper fornix was free from the new growth. The tarsal conjunctiva of the upper lid, in almost its entire extent, is covered with the new growth, which is here flatter and more compressed. The tumor ceases sharply at the conjunctival margin. The portion on the conjunctiva is more granular. There is a small isolated tumor near the cornea.

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[1] Elschnig, these ARCHIVES, No. 3, 1889.

[2] *L. c.*

October 29th, the tumor, together with the underlying conjunctiva, was extirpated, and the conjunctiva united by sutures. The wound healed by first intention, but caused a slight entropium of the upper lid. Nine months later a tumor 3 *mm* in diameter was found in the upper lid and removed. The conjunctiva was otherwise normal.

Case 2.—I. Pollack, æt. 68, admitted with senile cataract. The conjunctiva of the right lower lid and the lower half of the bulbar conjunctiva was somewhat injected. From the lower outer quadrant of the conjunctiva bulbi springs a lobular tumor the size of a hazel-nut, of soft consistence, rose-red color, and pervaded with numerous small vessels. The tumor hangs from a small pedicle and spreads out over a considerable area. When the lower lid is retracted the tumor projects through the palpebral tissue and is only returned with difficulty. It was first noticed a year before. The tumor was removed with the scissors and the wound healed rapidly.

HISTOLOGICAL CONSTITUTION.

In all the cases examined the connective-tissue framework was poorly developed in comparison with the epithelial strata. The connective tissue stroma was most marked in Case 1. It was of the ordinary type of fibrous tissue such as is seen in the connective-tissue portion of the conjunctiva. At many points it showed a reticular arrangement, and its meshes were filled with lymph corpuscles; in the individual papillary elevations of the tumor the connective tissue extended in parallel tracts, compressed in the pedicle and spreading out into a reticular structure in the papilla proper. In the smallest isolated portions of the tumor the connective-tissue tracts were irregularly arranged, and were composed of fine fibres and provided with a great number of blood-vessels, so that the connective tissue often presented itself as an adventitial sheath of the vessels. The epithelium covering the tumor made up its chief mass. Near the connective tissue it was mostly cylindrical in form, and above this the cells gradually assumed the pavement form.

Division by Fragmentation (J. ARNOLD).

Beside the mitotic figures which are seen in all stages, there are a series of figures which must be regarded as examples of Arnold's[1] type of direct fragmentation.

The variety of these forms observed and their irregularity warrant an accurate description. In all, the chromatic substance was degenerated into from two to six masses which stained well and showed no traces of fibrillar structure; the masses were often united, however, by fibrils, which stained well, or in case the fibrils were broken, the separate masses showed fibrillar processes. These forms were not seen frequently near the vessels, but were seen particularly in cells which lay in the superficial layers of the epithelium, among cells with unchanged nuclei and cells showing mitotic changes. The nuclei of the individual cells were of inconstant and irregular form. The nuclear membrane had always appeared and the structureless chromatic masses lay distributed through the substance of the cell.

After a very detailed description of karyokinesis according to modern views (which as belonging to general histological technology have been admitted in the translation), the author comes to the conclusion that the course of the mitotic division in the epithelial cells of papilloma is exactly of the type which is common in many of the lower vertebrates.

PAPILLOMA OF THE PALPEBRAL CONJUNCTIVA WITH PAPILLOMATOUS FORMATION OVER THE WHOLE SURFACE OF THE SKIN.

J. D., æt. 59, suffered in 1878 from hæmaptoë and an eruption on the skin with photophobia, lachrymation, swelling of the lids, and warty protuberances on the lids. In 1880 portions of the skin were covered with more or less prominent warty excrescences, partly pointed, partly bulbous. The limits of the growths were not sharply marked. The eyelids and the neighboring skin were thickly covered with pigmented papillomata the size of a hemp seed and smaller, projecting 2–3 *mm.* The conjunctiva was also covered with small warts, larger toward the lid margin. The lids were thickened, darkly pigmented, and slightly reddened ; the cilia were for the most part wanting ; the muco-purulent conjunctival secretion formed crusts on the lids.

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[1] Arnold, after extended investigations (see Waldeyer, *Arch. f. mikroscop. Anatomie*, vol. xxxii., 1888, p. 111), recognized the following forms of nucleus division: (1) Segmentation, direct and indirect; fragmentation, likewise direct and indirect. "If the nucleus split in an æquatorial or in a meridional plane, and the two parts, mostly of equal size, separate with an even surface, we have 'segmentation.' This is indirect when accompanied by karyokinetic changes, otherwise direct. In fragmentation the dividing surfaces of the daughter nucleus are irregular. Here also karyokinetic changes may be found (indirect fragmentation), or are wanting (direct fragmentation)."

Therapy : one per-cent. solution of salicylic acid. The patient lost weight, complained of pain in the stomach and pain on pressure in the pyloric region. A profuse diarrhœa came on ; the patient became worse, and died Jan. 10, 1881. The necropsy showed carcinoma of the posterior wall of the stomach, endarteritis of moderate degree, shrunken kidney, papilloma of skin, marasmus senilis.

Pieces of skin taken from the cadaver were hardened in absolute alcohol, and imbedded in celloidin. The upper lid was cut in sagittal sections and stained with carmine, safranin, and Ehrlich's hematoxylin and eosin. The preparations so obtained showed the skin and mucous membrane of the lids to be covered with tufted finely papillary warts. On the conjunctiva of the lid toward the fornix the papillæ were hypertrophied but not branched. The connective-tissue stroma of each papilla was composed of finely meshed fibrous tissue containing lymphoid cells, and corresponding to that of the broad papillæ which are found in the conjunctiva of individuals suffering from long-continued conjunctival catarrh. The epithelium is a many-layered pavement-epithelium, with an underlying layer of cylindrical cells.

All these formations showed on their surface the characteristics of the mucous membrane from which they sprang. At the free margin of the lid the individual papillomata took on an epidermoid character. Each presented on its surface a layer of cells corresponding to the epidermis, and a subjacent connective-tissue layer rich in vessels and nerves, corresponding to the corium. The epidermoidal portion increased in thickness from the free margin of the lid toward the outer surface, and was seen to consist of two layers, corresponding to the stratum corneum and the stratum germinativum of the true epidermis. The first layer is composed of several layers of flat scales without nuclei and taking the stain very faintly. The germinative layer, on the contrary, is composed in its deepest portion of a single layer of cylindrical cells resting immediately on the free surface of the sclera, while its superficial portion consists of polygonal prickle cells. The entire germinative layer is thickened, and the smaller papillomata are due to a proliferation of the cells of this layer. The mesoblastic portion of the individual elevations is composed of numerous conical formations—the papillæ corii. These are very vascular, contain numerous nerves, and pass over into a finely fibrillar non-fatty subcutaneous connective tissue, identical in histological structure with the connective-tissue basis of the normal skin of the lids. In the larger papillomata which show a marked development of the mesodermic connective-tissue layer, these subcapillary tracts of connective tissue have taken part in the structure of the individual excrescences. Some of the papillæ show new hair formation.

The ætiology of this case is not clear; no specific micro-organisms were found in the tissues. The effect of therapeutic remedies could not be studied, because of the lethal ending, which came soon after the patient's admission to the clinic.

I have found but one analogous case in the literature, reported by Lang.[1]

Explanation of the Figures on Plate XIV.

Fig. 1. Axial longitudinal section through a bud of the papilloma in Case 2. Slight development of the connective-tissue stroma, marked development of the epithelial covering. Ob. 2, Oc. 3. Reichert, tube drawn out.

Fig. 2. Sagittal section through the entire thickness of the upper lid of the case of general papillomatosis. Ob. 2, Oc. 3. Reichert, tube drawn out.

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[1] Lang. A case of papillomatosis universalis (from the dermatological clinic of Prof. Lang at Innsbruck).—*Vierteljahreschr. f. Dermatologie u. Syphilis*, 10. Jahrg., 1883, p. 337.

RETINITIS DIABETICA.

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(*With one Wood-cut.*)

R*ETINITIS diabetica* has hitherto been the name of retinal inflammations depending upon *diabetes*, without signifying any peculiar disease which could be recognized by its definite and characteristic appearance. The various forms in which it had been seen and described were considered very much the same as those of albuminuric retinitis. Thus Noyes, in his text-book,[1] published in 1890, says that "the features of both are to a great degree identical."

In Loring's work,[2] which has just been published, we find the statement that "the diabetic form of retinitis offers but little which is distinctive of presence of sugar in the urine. As a rule, however, there is more hemorrhage and less exudation or white plaques than in retinitis albuminurica, and the spots are similar and not so apt to be situated in the macula, or grouped in a zonular manner around the papilla." Gowers is scarcely more accurate. In the latest edition of his work on ophthalmoscopy[3] he describes the retinal changes as "bearing considerable resemblance to those of albuminuria, and still greater resemblance to those seen in some cases of pernicious anæmia. . . . White spots of degeneration are frequently present, commonly of moderate size, scattered over the fundus. . . . They differ from the patches of albuminuric retinitis in *shape*, having less tendency to assume a circular form ; in *color*, having a more dingy shade of white, and in *grouping*, the star around the macula being seldom seen, although there is a tendency for the spots to be arranged in the form of incomplete rings."

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[1] "Diseases of the Eye," p. 568.

[2] "Vol. ii., p. 152.

[3] Amer. edition of 1890, p. 228.

Other American and English authorities are much less definite in their descriptions, and German authorities are equally unsatisfactory.[1]

That characteristic differences do exist between diabetic retinitis and other forms of retinal inflammation was pointed out several years ago by Prof. Hirschberg,[2] of Berlin. In a more recent article[3] he demonstrates that there are typical forms of diabetic retinitis, which present characteristic appearances. The number of cases upon which he bases this study is very large, comprising twenty-five cases, " which is a greater number than has hitherto been published by any single observer." He distinguishes three kinds of retinal disease that occur in diabetic patients. " First, a very characteristic inflammation of the central part of the retina with small, bright spots, and usually fine spots of blood. Second, hemorrhages of the retina, with the consequent inflammatory and degenerative changes, including hemorrhages into the vitreous, hemorrhagic infarction of the retina, and hemorrhagic glaucoma. Third, rare forms of retinal inflammation and degeneration, whose connection with the constitutional disease yet remains to be demonstrated." There are, of course, intermediate forms, presenting the characteristic features of more than one type.

In the first class we find small, white, shining spots in the central part of the retina. These lie in the retinal tissue. In the macular region they are most marked, often forming little semicircles or curved streaks, with minute indentations along their margins. They may become confluent and form long streaks, though they rarely present the stellate form of albuminuric retinitis. A few bright spots may be seen scattered over the retina to the nasal side of the papilla. They never cause any disturbance of the retinal pigment. Fine hemorrhages may be visible throughout the region affected and beyond it, in the form of dots, small spots, and fine streaks. In these cases, the papilla is not altered either in its color or its margin; there is no diffuse haziness of the retina, nor are there any visible changes in the blood-vessels, such as variations in calibre. Large opacities of the vitreous are not found in this form. It is this type that is pathognomonic of diabetes. The disease occurs in persons of from forty-five to sixy-five years of age, who have commonly suffered with diabetes for a long time, often for several years, occasionally though to such a slight extent that their general disease remained undetected before the eyes became affected. It usually develops gradually, and always affects both eyes. The visual disturbance is due to central scotoma. Vision ranges from $\frac{1}{2}$ or $\frac{1}{3}$ to $\frac{1}{10}$ or $\frac{1}{20}$, but not to complete blindness. It may improve slightly, but usually will either remain stationary or progressively diminish. There may be

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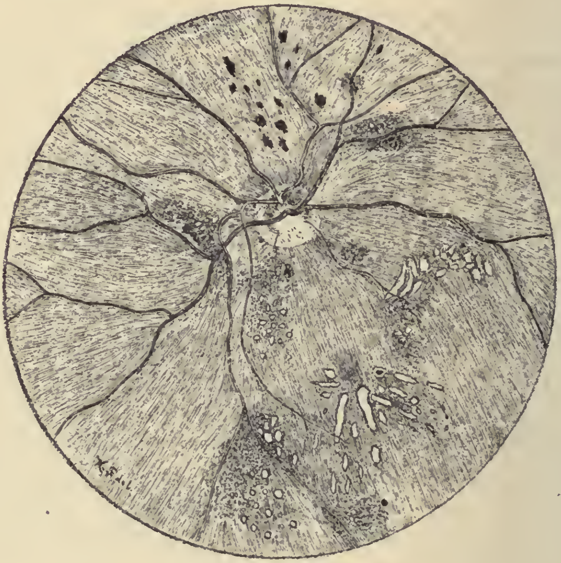
[1] See Hirschberg, *Deutsche med. Wochenschr.*, 1890, No. 51.

[2] " Sehstörungen durch Zuckerharnruhr," *Deutsche med. Wochenschr.*, Nos. 17 and 19, 1887.

[3] " Ueber diabet. Netzhautentzuendung," *Deutsche med. Wochenschr.*, 1890, Nos. 51 and 52.



RETINITIS DIABETICA.

lenticular opacities, and the patient may have paresis of the accommodation at the same time.

Hirschberg believes that these white spots owe their origin to changes in the finest blood-vessels; as mentioned above, the larger vessels appear normal. Their histology is not known.

During the last year a case quite typical of this form came under my observation, and the patient, a lady sixty-four years of age, who had been suffering with diabetes for several years, it having been looked for and detected by her family physician as the cause of pruritus vulvæ of long standing. Albumen has never been found in the urine. Her general condition had not suffered much during these years, though the patient had lost some weight. Her vision had become affected within two to three years, and was found to be: L E, + 2.5 D sph V $\frac{5}{10}$; R E, + 2.75 D sph V $\frac{5}{8}$ (June 16, 1890), the impairment being due to central scotoma, as is always the case in this form. The ophthalmoscopic examination shows a few peripheral striæ in both lenses, but otherwise the media were perfectly clear. The retina presented the typical form of diabetic retinitis described above, and shown in the drawing of the upright image of the retina of the left eye, which was slightly more affected than the right. There are the intensely bright, white spots arranged around the macula in streaks and lines, and, perhaps, approach the stellate figure more than is commonly the case. There are other spots of degeneration, less marked and finer in form, farther from the macula, though few reach beyond the papilla. Scattered hemorrhagic spots are seen among the white spots and to the nasal side of the papilla. The papilla is quite clear, of normal color and sharply defined. There were no abnormal variations in the calibre of vessels.

I have not been able to repeat the examination since then, but have learnt that her vision has become slightly worse. The treatment has been purely dietetic.

A CASE OF PULSATING EXOPHTHALMOS.

By P. B. WING, M.D., Tacoma, Washington.

Patient, a young man, age about 22, occupation laborer. In August, 1889, he fell some distance, striking on his head, was taken up in an unconscious condition and carried to the hospital. He remained in this condition for twenty-four hours and was in the hospital about ten days, and was discharged apparently well.

In December following, about five months after the injury, he noticed that the right eye began to protrude ; it progressed slowly until the following June, when his physician, Dr. Beardsly, came with him to my office. At that time the eye protruded so much that the lids could with difficulty be closed over it. The conjunctival vessels were large and tortuous. Vision about $\frac{20}{200}$. There was a distinctly pulsating tumor at the upper and inner angle of the orbit. There was not at that time and never had been any pain or soreness. By pressure the eye could be made to partly recede. Pressure upon the common carotid artery caused the pulsations to cease.

My diagnosis was pulsating exophthalmos, and I advised ligation of the common carotid as the operation offering the best results. I have no doubt that there had been a fracture at the base, passing across the cavernous sinus and causing a communication to exist between it and the internal carotid artery, thereby forcing arterial blood directly into the ophthalmic veins, causing the great distension and strong pulsation ; in fact, it was a tumor composed of pulsating varicose ophthalmic veins.

The patient was again sent to the hospital and the common carotid ligated by Drs. Beardsly and Wintermute. The pulsations stopped at once and never since returned. The eye began to gradually sink back into its place, and to-day, fourteen months since the operation, the protrusion is not noticeable except upon very close observation. The vision is $\frac{20}{40}$ +. Patient has been at hard labor for the past eight or nine months.

I have reported this case for three reasons—it is the only one I have ever seen, it made a fine recovery, and because I want some well-informed brother[1] to give us some statistics in regard to its frequency and the results obtained by the various operations for its relief.

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[1] The fullest description of pulsating exophthalmos, with tabulated records of 106 cases, is by H. Sattler in Graefe-Saemisch's "Handbook of Ophthalmology," vol. vi., pp. 745 to 1074. One of the most brilliant cases is that by Dr. E. Grüning, described in these ARCH., vol. v., p. 40, in which both eyes were protruding and totally blind. Perfect recovery with immediate restoration of sight by ligation of the left common carotid. In a case of the writer's, a temporary cure was effected by ligation of the carotid, a permanent cure (of the relapse) by extirpation of the aneurismal varix of the orbit (these ARCH., vol. xii., p. 201). Ligation of the carotid cures the majority of cases.—H. K.

PRESSURE-INUNCTION IN THE TREATMENT OF CERTAIN CHRONIC HYPERPLASTIC CONDITIONS OF THE EYE.

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I HAVE ventured to propose the term *pressure-inunction* to describe a form of treatment involving mercurial inunction combined with more or less continuous pressure.

The analogy presented by a chronic inflammatory hyperplasia of the synovial and fibro-cartilaginous structures of the knee-joint to a chronic hyperplastic condition of the cornea, sclera, and vitreous body suggested to my mind the idea that, if mercurials combined with pressure could be of service in the one case, it might also be found useful in the other. I would urge in support of this contention that the selective power of drugs is well known, and that it is undeniable that certain diseases prefer certain tissues as a local habitation. I would further point out the developmental relationship between cartilage, fibrous tissue, sclera, and conjunctiva.[1] Are they not all derived from the mesoblast? and is it not admitted that a pathological relationship exists between tissues of the same developmental origin?

Arguing on these premises I have for many years carried on a series of observations, and have had ointments of various strengths and of different preparations of mercury compounded, and have arrived at a standard ointment made of

Calomel	gr. xxx.
Vaseline	℥ i.

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[1] "Eierstock und Ei," Waldeyer; "Entwicklungsgeschichte des Menschen," Kölliker.

very carefully rubbed up. It is applied freely to the closed eyelids, and, in cases of corneal opacity, a small piece is inserted in the conjunctival sac. A pad of absorbent cotton is then applied and over it three or four turns of a good elastic flannel bandage. This dressing is worn from two to three hours twice daily.

The cases in which I have found most benefit from this treatment are those of corneal opacities of a dense type, interstitial deposit, plastic deposit on the lens from iritis, opacities of the vitreous, and some obstinate cases of scleritis. The improvement is slow and the treatment must be persisted in for some months. I have never seen mercurialization from this treatment. It will be found that some persons will bear a stronger ointment than the standard one, in others it causes too much irritation and must be weakened or intermitted. I have occasionally injected five minims of a 2-per-cent. solution of common salt beneath the ocular conjunctiva after the method of Brudenel Carter, and have intermitted pressure-inunction for a few days. The hypodermic injection of sodium chloride should not be repeated oftener than once in three or four days, and the initial dose should not exceed two drops.

While this method of treatment is by no means infallible, I still have been able to obtain results which I would not have hoped for under other circumstances.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FOURTH QUARTER OF THE YEAR 1890.

BY ST. BERNHEIMER, HEIDELBERG; C. HORSTMANN AND P. SILEX, BERLIN.

WITH THE CO-OPERATION OF

S. M. BURNETT, Washington; DANTONE, Rome; HIRSCHMANN, Charkow; E. MARCKWORT, Antwerp; P. V. MITTELSTÄDT, Metz; L. WERNER, Dublin; C. H. A. WESTHOFF, Amsterdam; SCHIÖTZ and OLE BULL, Christiania, etc.

Translated by Dr. WARD A. HOLDEN, New York.

Sections I.–V. Reviewed by DR. ST. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TEXT-BOOKS, MONOGRAPHS, TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS.

405. NORTON, GEO. S. An analysis of 1,022 eyes examined under atropine. *Jour. Ophth., Otol., and Laryngol.*, Oct., 1890.

406. SUAREZ DE MENDOZA. L'audition colorée. *Soc. d'Opht. de Paris*, No. 4, 1890.

407. SEBRENIKOWA, FRAU. Ophthalmological observations at the government hospital in Perm, during the year 1889. *Wjestnik Ophth.* No. 6, 1890.

408. MOTSCHULSKY. A case of complete cyclopia. *Russkaja medic.*, No. 43, 1890. (Description of a preparation in spirits belonging to the Society of Physicians in Kertsch.)

409. HAAB, O. Pathological anatomy of the eye. Ziegler's "Text-book of Special Pathological Anatomy," sixth edition.

410. SCHWERSENSKI, G. On the changes in the periphery of the fundus. *Inaug. Dissert.*, Leipsic.

411. LORENZ. On the proper construction of clinics. *Centralbl. der Bauverwaltung*, J. 1889-1890, Berlin.

NORTON (405) tabulates carefully 1,022 eyes examined under atropine. He finds that the axis of the cylinder in astigmatism sometimes changes under atropine, in some instances as much as 45°, showing an unequal contraction of the ciliary muscle. Since he has been using the ophthalmometer he finds the use of atropine less necessary. As a guide in general to the use of atropine, he takes the fact that the glasses which have been ordered without atropine are unsatisfactory. BURNETT.

SEBRENIKOWA (407) reports 3,055 cases with 780 operations, 126 being extractions, 48 without iridectomy. She does not favor the latter method of operating. HIRSCHMANN.

HAAB (409), in his pathological anatomy of the eye, gives in a condensed form a general and very accurate description of the most important pathological changes. The careful drawings are of value, as is also the bibliography at the end of the chapters.

SCHWERSENSKI (410) in his dissertation spoke of the frequence with which the peripheric changes in the fundus, described by Magnus, were observed, and of the relative number of these cases which were to be ascribed to old age, sex, refraction, and inflammation. In general he often found these changes to have a certain etiological connection with senescence and with inflammation ; he regarded them also as characteristic for myopia, and in this case probably due to mechanical causes, such as stretching and tearing. He could not speak definitely as to their relation to hyperopia.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

412. DE SCHWEINITZ, G. A case in which the effect of homatropine upon the accommodation was unusually prolonged. *Ophth. Rev.*, vol. ix., p. 353.

413. MADDOX, E. The investigation by the rod-test of paresis and paralysis of the ocular muscles.

414. BERRY, G. Note on the metre-angle in latent and manifest muscular deviation. *Trans. Ophth. Soc. of the United Kingdom*, vol. x., p. 190.

415. BISSELL, E. F. Note on the shadow-test. *Jour. of Ophth., Otol., and Laryngol.*, Oct., 1890.

416. Koller, Carl. Elimination of the cornea and the effect upon the refraction of the eyes. *Amer. Jour. of Ophth.*, No. 7, 1890.

417. Simi. Posterior sclerotomy. *Boll. d'ocul.*, vol. xii., p. 22.

417a. Jays. Mesure de la convergence dans la vision binoculaire. Choix d'unité de convergence. Série métrique des prisms en oculistique. *Soc. d'Opht. de Paris*, Oct., 1890.

418. Leroy. Influence de la distance de l'observateur dans la mesure des amétropies à l'image droite. *Rev. génér. d'opht.*, No. 10, p. 433, 1890.

419. Dransart. De la suspension dans le nystagmus des mineurs et la neuro-retinite. *Jour. d'ocul. du Nord de la France*, p. 38, 1890.

420. De Wecker. Résection et arrachement du nerf optique. *Soc. d'Opht. de Paris*, 1890.

421. Buchner, H. The chemical irritability of leucocytes and their relation to inflammation and suppuration. *Berliner klin. Wochenschr.*, No. 47.

In De Schweinitz's (412), case after using homatropine twice, the accommodation was not wholly restored at the sixth day.

Werner.

Maddox (413) describes his glass-rod test in detail. He uses with it a scale either in degrees or in metres. With this test latent divergence may be shown, which could not be discovered in any other manner. Werner.

Berry (414) found the actual association between accommodation and convergence in cases of squints to be given by the measurement of the metre-angle, the half of the angle of deviation being taken. Werner.

Bissell (415) has used the shadow-test for some time and testifies to its utility. It is easy to use, and by it results may be obtained where other methods fail.

After a detailed description of the optical phenomenon of Bellarminoff's method of making the fundus visible by eliminating the refraction of the cornea, Koller (416) described his modification of this method. In place of a plane glass plate, he uses a glass shell like an artificial eye, but with the convex outer surface ground plane; the concave inner surface lies in contact with the cornea. The rays emerge strongly divergent from this apparatus, and the illuminated fundus is seen with the naked eye.

Burnett.

Simi (417) made repeated sclerotomies at the equator of the ball in a case of glaucoma and in one of traumatic panophthalmitis. No disturbances attributable to the operation occurred, and the author therefore commends the study of it to his associates. The equatorial sclerotomy checked the glaucoma, where a deep corneal ulcer and peri-keratic swelling prevented him from performing an iridectomy or sclerotomy at the sclero-corneal margin. In the case of panophthalmitis the small operation immediately relieved the pain caused by the increased tension.

Dantone.

Dransart (419) tried suspension in cases of atrophy of the optic nerves without success. On the contrary, he obtained favorable results with this treatment in cases of neuro-retinitis hemorrhagica and in cases of miner's nystagmus.

Marckwort.

With the purpose of limiting enucleation to cases of intraocular tumors, De Wecker (420) experimented on the cadaver in order to find whether it were possible to tear the optic nerve loose near the optic foramen in place of cutting it. He found the operation impossible.

Marckwort.

Buchner (421) thoroughly investigated the emigration of leucocytes under chemical influences. Various vegetable and animal substances affect the leucocytes in different manners. He studied: (1) The irritation exerted on leucocytes by bacterial albumens (pneumo-bacillus protein), obtained by the digestion of bacteria masses with 0.5 per cent. potash lye. The substance was sealed in small glass tubes, sterilized, introduced under the skin, and the point broken ; it was found to be very active. (2) The irritation of products of decomposition. Glycocoll and leucin alone showed the slightest chemical action. (3) Vegetable caseine, gluten caseine, weakly alkaline, and legumin exerted a strong attraction on the leucocytes. Starch-flour acted negatively. (4) Transformation products of animal tissue. Albumen-peptone acted negatively, cement substance, on the contrary, actively. Muscle, liver, and kidney of the rabbit in 5- to 10-per-cent. solution showed a strong attraction. (5) Direct leucocytosis by chemical irritants. In the rabbit all substances which irritate the leucocytes, when injected in the aural veins, caused an increase in leucocytes. The ratio increased from 1:318 to 1:38. (6) The relation of the leucocytic attraction to inflammation and suppuration. This attraction of the leucocytes cannot be differentiated experimentally from the inflammatory irritation of the fixed tissue-elements. Extensive experiments with the bacillus pyocyanus were undertaken in men.

III.—INSTRUMENTS AND REMEDIES.

422. Simi. On hot ocular douches. *Boll. d'ocul.*, vol. xii., p. 23.

423. De Wecker and Masselon. Sur les montures de lunettes. Un ophtalmo-statomètre. *Ann. d'ocul.*, vol. civ., p. 147.

424. Redmond, D. An electro-magnetic rotating needle for the discission of tough and calcareous secondary cataracts.

Simi (422) commends ocular douches with hot sublimate solutions ($\frac{1}{3}\%$ and from 36–45° C.). He has had an irrigator constructed which keeps the solution at the temperature desired.

Dantone.

De Wecker and Masselon (423) speak of the necessity of ordering not only proper glasses, but also properly fitting frames. If the spectacles are to be made by an optician who cannot see the patient, besides the pupillary distance, the height of the bridge of the nose should be measured, the breadth of the cheeks, and the distance of the ears from the eyes. For the measurement of the bridge of the nose the authors take a base-line which passes through both pupils when the eyes are directed straight forward. The authors have had two particular instruments made for this purpose.

Marckwort.

IV.—ANATOMY.

425. Antonelli. A contribution to the study of the morphological significance and the structure of the ciliary ganglion. *Giorn. dell' Assoc. dei naturalisti e med. di Napoli*, vol. i.

426. Nicati, W. La glande de l'humeur aqueuse, glande des procès ciliaires ou glande uvée. *Arch. d'opht.*, vol. x., No. 6, 1890.

Antonelli (425), on the ground of extensive comparative anatomical and histological researches, considers the ciliary ganglion as belonging to the oculomotor nerve, and the roots of the trigeminus and sympathetic as accessory.

Dantone.

Nicati's (426) investigations treat of the apparatus secreting the aqueous humor in its histological, physiological, and pathological relations. He calls by the name "glande de l'humeur aqueuse" the portion of the uveal tract secreting this fluid, particularly the chorio-capillaris and the ciliary processes which form the secreting surface. He reviews the older and the newer ideas as to the seat of the secretion, nerve influence upon the intraocular tension, quantity, composition, and fluorescine coloring of the aqueous, and then treats of the histological relations of the "glande uvée" and its adnexa. He describes (1) an epithelium (outer or pigment layer, inner layer, or that composed of the cylindrical cells of the cilary processes); (2) fibres connecting the epithelium of the processes with the lens (in the eye of the rabbit, dog, and hen), representing the peculiar suspensory apparatus of the lens, independent of the posterior wall of the canal of Petit, which latter is formed of the hyaloid membrane with its connective-tissue reinforcements; (3) a capillary network—the chorio-capillaris anterior and posterior to the ora serrata, bounded inwardly by the lamina vitrea, outwardly by Sattler's membrane, and for this reason able to secrete fluid only anteriorly on the surface of the processes where the lamina vitrea ceases to be a continuous layer. The chorio-capillaris is a secretory organ and has nothing to do with the nutrition of the retina. (4) Arteries and veins (the ciliary vessels). (5) Muscle (smooth muscular fibres in the entire extent of the choroid and the ciliary muscle). (6) Nervous system with sensory motor and sympathetic fibres. (7) Orifices of the glands: the system of spaces of the suspensory ligament of the lens, the posterior chamber, and the communication between them and the spaces between the ciliary processes. (8) A receiving reservoir (the anterior chamber) and the excretory ducts: the iris and the spaces of Fontana, the crypts and stomata of the iris, and the lymph-vessels and perivenous lymph-spaces, which lead to the anterior and posterior ciliary veins.

V. MITTELSTÄDT.

V.—PHYSIOLOGY.

427. PIERINI. Discussion of some disputed points in physiological optics. *Giorn. della R. Accad. di Med. di Torino*, 11–12.

428. SEGAL, S. L. On the phosphenes which are seen at the point of pressure. Read before the Society of Physicians in Odessa. *Wratsch*, No. 48, 1890.

429. SCHIRMER, O. Does Weber's law hold good for the light sense? *Graefe's Archiv*, vol. xxxvi., 4, p. 121.

430. JERUSALEM, W. Laura Bridgman. The training of a blind deaf-mute. A physiological study. *Zeitschr. f. Psych. u. Phys. d. Sinnesorg.*, vol. i., part 6, p. 505.

431. Raehlmann. Physiologico-pyschological studies on the development of the faculty of recognizing faces in children, and in patients born blind who have been operated upon. *Zeitschr. f. Psych. u. Phys. d. Sinnesorganen*, vol. ii.

Pierini (427) criticises, apparently justly, the views on the position of the centre of rotation of the eye, which Albertotti held in constructing his autoperimeter, and which Landolt held in the investigations with his chiastometer. Secondi's opinion, that the angle α changes in different positions of the visual lines while the accommodation remains the same, is also considered untenable by the author. Dantone.

Besides the well-known pressure phosphene opposite the point of pressure, Segal (428) has observed a second less intense phosphene in the direction of the point of pressure, and especially marked when the pressure is intermittent. The author explains this as caused by the conduction of the pressure through the vitreous to the retina opposite. Segal saw the phosphene directly before him when intermittent pressure was made on the cornea. Hirschmann.

Schirmer (429) sought to establish the truth of Weber's law for the light sense by a careful study of adaptation within certain limits of illumination. The experiments of Aubert and Helmholtz, which apparently disproved the law, were criticised, and by a series of original experiments the author attempted to prove :

(1) That Weber's law of the least perceptible differences holds good for the light sense within the limits of illumination 1-1000 M K, if the eye is permitted to exercise its complete power of adaptation ; the truth of the law is therefore dependent upon certain physiological conditions.

(2) The truth of Weber's law can be explained only by means of the physiological process of adaptation.

(3) In the normal eye the adaptation does not, or at least not always, proceed regularly in the dusk when the daylight decreases.

Sections VI.–XI. Reviewed by Dr. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

430. Weiss, L. Contributions to the anatomy of the orbit. iii.—On the relation of the orbit to the various forms of the head and face. Tübingen, 1890. H. Laupp.

431. BERRY, G. On myopia. *Ophth. Rev.*, vol. ix., p. 327.

431a. P. SMITH. A reply to the preceding. *Ibid.*, p. 345.

432. KROTOSCHIN. Anatomical contributions regarding the development of myopia. *Arch. f. Augenheilk.*, vol. xxii., p. 393.

433. JONG, W. de. A contribution to the development of myopia. Examination of the eyes of 3,930 school children in Leyden. *Zeitschr. f. Schulgesundheitspflege*, 1890, No. 12, p. 739.

434. KOTELMANN. Dr. Francis Warner's report on the examination of fourteen London schools. *Ibid.*, No. 12, p. 697.

435. GALEZOWSKI. A study of some grave varieties of myopia and the means of curing them. *Rec. d'opht.*, 1890, p. 510 and 571.

436. MARTIN, G. Astigmatic amblyopia. *Ann. d'ocul.*, vol. civ., p. 101.

437. KOLLER, C. On the determination of astigmatism with the ophthalmometer (Javal-Schiötz). *Jour. Amer. Med. Assoc.*, Sept. 13, 1890.

438. GALEZOWSKI. On the diagnosis of irregular astigmatism and its correction by means of cylindrico-conical lenses.

The view is held in many quarters that the conformation of the orbit has an influence on the form of the eyeball, and consequently on its refractive condition. WEISS (430), in order to test this relation, reports accurate measurements made on more than one hundred skulls.

BERRY (431) states that benign myopia differs ætiologically and anatomically from myopia with affection of the choroid. The worst cases of the latter category are found in the uneducated classes, and are more frequently seen in the clinic than in private practice. It is not proven that convergence increases the sagittal diameter of the ball, and there are grounds for opposing this view. WERNER.

P. SMITH (431a) replies by giving statistics of 4,000 cases of myopia. In 2,000 private cases 64 had myopia over 10°. While in 2,000 hospital patients only 27 had myopia over 10° (7 of these had 20° or more, while 8 of the former only reached this; this is in favor of Berry's contention). There is no definable boundary between benign and malignant myopia, and it is difficult to prove it. In a hypermetropic eye the convergence does not cause the same strain on the sclera (as shown by Emmert). In unilateral myopia the effort of convergence is made, but is impeded by the shape of the eyeball. WERNER.

KROTOSCHIN (432), after making a great number of measurements, accepts Stilling's theory, that a low orbit disposes to myopia, and a high one to emmetropia or hyperopia.

GALEZOWSKI (435) speaks of myopia in general. According to him, myopia almost always remains stationary when accompanied with astigmatism. Myopia sometimes develops at the age of 2–3 years. Even in low degrees of myopia glasses should be worn for near work. MARCKWORT.

MARTIN (436) publishes another long paper, in which he reports much new clinical matter, which serves to support his idea of astigmatic amblyopia. By astigmatic amblyopia the author understands an amblyopia which, as a result of astigmatism, develops in one meridian. A tabular report is given of the condition of 108 eyes with astigmatic amblyopia. When a considerable astigmatism develops from a trauma in that period of life before the retina has completely gained its functions, no astigmatic amblyopia occurs. When an astigmatic accommodation corrects a corneal astigmatism, no amblyopia develops. There is not so great a tendency to this correction when the other eye is not astigmatic and has good vision ; therefore the amblyopia is usually more marked in cases of monocular astigmatism than in cases of binocular. The relation of objective to subjective astigmatism is also discussed and illustrated with statistics. In 108 cases, the objective and subjective astigmatism were equal in 49. In myopes the subjective astigmatism is often greater than the objective, while the contrary relation is found in simple hyperopic astigmatism. Martin finds the different acuteness of vision at the different axes, and designates one the maximum acuteness and the other the minimum acuteness of vision. The acuteness of vision usually found corresponds nearer to the maximal meridian acuteness than to the minimum meridian acuteness. MARCKWORT.

GALEZOWSKI (438) has had cylindrico-conical lenses made to correct irregular astigmatism, and especially irregular lenticular astigmatism. While ordinary cylindrical lenses are segments of the surface of a cylinder, the new lenses represent portions of the surface of a cone taken parallel to its axis, and so having a different curvature at different distances from the apex of the cone. MARCKWORT.

VII.—LIDS.

439. Friedenwald, H. Cases of phlegmonous gangrene of the lid. *Amer. Jour. of Ophth.*, vol. vii., p. 285.

440. Hallopeau. Hydro-adenoma of the lid, complicated with epithelioma. *Union méd.*, 1891, No. 4.

441. Amorin. On the restoration of the lids by skin-grafting. *Thèse de Paris*, 1890.

442. Fontan. Blepharoplasty by scissor-shaped flaps for the total restoration of both lids. *Soc. d'opht.*, Nov. 4, 1890.

443. Ray, J. M. Report of a case of transplantation without a pedicle for cicatricial ectropion. *Jour. Amer. Med. Assoc.*, Oct. 11, 1890.

444. Robertson, A. Note on the treatment of severe senile entropium. *Edinburgh. Med. Jour.*, 1890, No. 1526, p. 497.

445. Harlan, G. C. New operation for symblepharon. *Ophth. Rev.*, vol. ix., p. 351.

446. Snellen, H. New method of treating symblepharon. *Trans. Ophth. Soc.*, vol. x. p. 207.

447. Scheffels, Otto. On advancement of the levator tendon. *Graefe's Archiv*, vol. xxxvi., 4, p. 374.

448. Browning, F. W. Associated contraction of the levatores palpebrarum superiorum with the internal recti. *Trans. Ophth. Soc.*, vol. x., p. 187.

Friedenwald's (439) case was that of a little girl who scratched herself on the temple. In the course of a few hours great swelling set in, followed by a gangrene and sloughing of almost the entire upper lid. The ciliary margin, however, was not involved. Eyeball not affected. Treatment constitutional, and local application of iodoform. Burnett.

Fontan (442) made a plastic operation in a patient, the skin of whose face had been extensively destroyed by carcinoma. There were wanting both left eyelids, the outer half of the left eyebrow, the outer portion of the forehead, the temple, and the cheek on the left side. A forehead flap was taken for the lower lid, and a nasal flap for the upper. Since the flaps crossed each other, like a pair of scissors, he called the operation blepharoplasty with scissor-shaped flaps. Marckwort.

Ray's (443) case was an ectropion of the upper lid, and the raw surface left by its detachment was 2 by 1½ inches. This was covered by integument taken from the arm, according to Wolfe's method, and kept in place by gold-beaters' skin and non-contractile collodion. Union was perfect. Three months after, the flap had shrunken to 1 by ¾ inch, but the lid covered the cornea nicely. BURNETT.

In symblepharon of the lower lid, HARLAN (445) dissects up the adhesion and turns in a flap of skin through an incision in the lid ; the flap having its raw surface in contact with the inner surface of the lid, and its sound surface toward the eyeball. WERNER.

SNELLEN (446), in bad cases of symblepharon, passes a pediculated skin flap in through a button-hole in the lid, the skin being in contact with the eye. His object in ptosis is to shorten the levator tendon by sutures, without cutting the skin. He recommends subconjunctival injection of sublimate 1:5000 for episcleritis. WERNER.

For entropium and trichiasis of the upper lid, SCHEFFELS (447) recommends the advancement of the levator tendon. This produces a secure cicatricial attachment between the fibres of the orbicularis on one side and the upper tarsal margin, with the fascia and tendon of the levator on the other, while at the same time advancing the tendon and changing the direction of its point of attachment.

In BROWNING'S (448) patient, with the action of the interna-rectus an elevation of the upper lid of the same side was observed, with convergence a similar elevation of both upper lids, and on looking downward the lids reached only to the horizontal meridian. WERNER.

VIII.—LACHRYMAL APPARATUS.

449. CAUDRON. Two cases of acute dacryo-adenitis. *Soc. d'Opht. de Paris*, Nov. 7, 1890.

450. SIMI. A case of abscess of the sac cured with anilin oil. *Boll. d'ocul.*, vol. xii., 24.

CAUDRON (449) reports two new cases of inflammation of the lachrymal gland. In the first case the swelling of the gland recurred at the menstrual periods, becoming each time more severe. The patient was seen but once. In the second case Meyer made a puncture through the conjunctiva and freed a clear fluid with good results. MARCKWORT.

Simi (450) reports another case of abscess of the sac in which anilin oil (8 drops to 100 gr. water) acted as a valuable antiseptic.

Dantone.

IX.—MUSCLES AND NERVES.

451. Berry, E. Note on the immediate effect of tenotomy on the concomitancy of a squint. *Trans. Ophth. Soc.*, vol. x., p. 202.

452. McAchran. Laceration of the internal rectus. *Amer. Jour. of Ophth.*, vol. vii., 11, p. 364.

453. Landolt, E. On muscular asthenopia. *Arch. d'opht.*, 1890, No. 6, p. 509.

454. Roosa, St. J. Muscular asthenopia. *Ophth. Rev.*, vol. ix., p. 282.

455. Webster, D. A case of paralysis of the superior oblique cured by tenotomy of the superior rectus of the same side. *Med. Rec.*, Jan. 7, 1890.

456. Raymond, P. A case of nuclear ophthalmoplegia externa. *Gaz. des hôp.*, 1890, No. 126, p. 1167.

457. Morel, F. A contribution to the study of ophthalmoplegia externa. *Thèse de Paris*, 1890.

458. Bouveret et Curtillet. Bilateral ophthalmoplegia externa of embolic origin. *Gaz. hebd.*, 1890, No. 40, p. 512.

459. Dehove. Bilateral paralysis of the third nerve, of hystero-traumatic origin. *Sem. méd.*, 1890, p. 455.

460. Guende. The muscular paralyses of the eye. *Rec. d'opht.*, 1890, No. 12, p. 705.

461. Romano-Catania. On a form of paralysis of the ocular muscles accompanied by myopia.

462. Christ. On the ætiology of nuclear ophthalmoplegia. *Deutsches Arch. f. klin. Med.*, vol. xlvi., p. 496.

463. Landolt, E. Ocular torticollis. *Bull. méd.*, 1890, p. 573.

464. Eversbusch, O. A case of traumatic ciliary neuralgia cured by the galvano-cautery. *Münchener med. Wochenschr.*, 1890, No. 51.

465. Allport, F. Some remarks on the relief of remote neuroses by the restoration of ocular equilibrium. *Amer. Jour. of Ophth.*, vol. vii., 11, p. 367.

Berry (451) measures the convergence not alone when fixing in the median line, but also about 30° to either side. The results

will only be the same when the concomitancy of the eyes is complete. He finds that the squint remaining after a tenotomy increases in amount on fixing toward the side of the squinting eye.
WERNER.

LANDOLT (453) objects to the opinion expressed at the Berlin Medical Congress that muscular asthenopia does not exist. He gives a brief description of the symptoms, and reports some observations respecting treatment which go to show, as he has previously stated, the advantage which advancement has over tenotomy.
V. MITTELSTÄDT.

ROOSA (454) is of the opinion that insufficiency of the ocular muscles is usually a consequence of an error of refraction; his reasons for coming to this conclusion being that he had always found prisms unsatisfactory, and that the correction of astigmatism removed the symptoms and the necessity for prisms. Tests for insufficiency, etc., are delusive owing to the varying strength of the muscles, and equilibrium exists only in a moderate percentage of people who are not asthenopic. The author admits the existence of *neurotic* asthenopia, which may not be relieved by correction of refractive errors, but in these cases tenotomies and prisms are only means of treating these cases by suggestion.
WERNER.

The paralysis of the superior oblique in WEBSTER'S (455) case was syphilitic, but did not yield to active anti-syphilitic treatment. A tenotomy of the superior rectus resulted in an over-correction, which was remedied later by an advancement of the same muscle. The cure was complete, there being no diplopia. BURNETT.

ROMANO-CATANIA (461) reports a case of congenital bilateral paralysis of the muscles supplied by the third and sixth nerves, excepting the sphincter pupillæ and the muscle of accommodation, in a man who was excessively myopic. Since this was apparently due to a functional disturbance of the nuclei of origin of the nerves in question, which had developed in fœtal life, the author, on account of the non-disturbance of innervation of the interior muscles of the eye, endeavors to support the opinion of many others (Hensen and Völkers, Kahler and Pick), according to whom the nucleus of origin of the third nerve has an extent of some 10 *mm*, through which are distributed the individual nuclei of the branches supplying particular muscles. In the case in question the middle and posterior portions of the nuclei are those at fault, since the nuclei for the interior muscles of the eye lie in the anterior portion. The symmetrical limitation of the disturbance may be anatomically explained by the supposition that the nucleus is supplied by several small arteries which do not anastomose (terminal arteries); a disturbance in the circulation of one or more of these terminal arteries would therefore cause a disturbance in the particular portion of the nucleus supplied. Similar cases have been described by Schroeder and Rählmann. The extreme myopia in this case was explained by the author as being due to the pressure exerted by the superior oblique, which muscle alone preserved its function, and which plays an important rôle in the development of myopia in general and especially when the vertical diameter of the orbit is short. DANTONE.

CHRIST (462) showed a complete paralysis of the fourth nerve and an incomplete paralysis of the third in both sides. At the autopsy a gliosarcoma was found involving the pineal gland, the region of the corpora quadrigemina, and the posterior portion of the third ventricle.

LANDOLT (463) described two cases of torticollis after paralysis of the fourth nerve. In order to correct the disturbing double vision the patient had brought his head into this peculiar position. The head was inclined toward the shoulder and the face turned to the opposite side. Tenotomy was advised but refused.

MARCKWORT.

EVERSBUSCH (464) reported a case of injury to the eye with a flax comb. The vision became normal, but there was pain in accommodation. A small cicatrix was seen at the corneo-scleral margin. When this was destroyed with the galvano-cautery, all difficulty was relieved.

ALLPORT (465) believes that properly performed tenotomies of the external muscles will in many cases relieve headaches, but does not think that they can affect remote neuroses, such as chorea, epilepsy, etc. BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

466. SNELL, S. Acute cellulitis of orbit; fatal result. *Trans. Ophth. Soc.*, vol. x., p. 51.

467. DRANSART. Symptomatologie, diagnostique, étiologie et traitement des hydrarthroses orbito-oculaires séreuses. *Jour. d'ocul. du Nord de la France*, 1890, No. 1, p. 6.

468. SPENCER WATSON. A case of osteoma of the infra-superciliary region, with the specimens removed by operation. *Trans. Ophth. Soc.*, vol. xi., p. 44.

469. Rockliffe, W. C. Case of cephalic tetanus following penetrating wound of the orbit; recovery. *Trans. Ophth. Soc.*, vol. x., p. 46.

Snell's (466) patient, a girl of 14, developed cellulitis in the right orbit as the result of diseased molar teeth which had caused slight necrosis of the jaw. She died of coma in spite of the local improvement which resulted from treatment. Werner.

Dransart (467) believes that a serous effusion may collect beneath Tenon's capsule, and, by pressure on the optic nerve, lead to its atrophy. This exudation may be allowed to escape by means of an operation suggested by Dransart. Capsulotomy is also indicated in cases of phlegmonous tenonitis. In various other diseases Dransart makes a capsulectomy. Marckwort.

Watson (468) removed a small ivory osteoma from a woman æt. 30. It lay quite free in the subcutaneous tissue of the right eyebrow. Werner.

In Rockliffe's (469) case, a boy æt. 7, injury to the left eyebrow was followed by ptosis and facial paralysis, accompanied by trismus and slight pharyngeal and respiratory spasms. Recovery took place in fourteen days. Werner.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

470. Andrews, J. A. Purulent ophthalmia; the ætiology and treatment of the disease. *New York Med. Jour.*, vol. lii., No. 25, p. 681.

471. Schmidt, E. Report of the eye department of the Marine Hospital in Sebastopol for the year 1889. *Med. supplement to the Moskoi Sbornik*, Oct., 1890.

472. Darier. The surgical treatment of granular conjunctivitis. *Soc. d'Opht. de Paris*, Dec. 2, 1890.

473. Reid, T. Lymphoma of the conjunctiva. *Trans. Ophth. Soc.*, vol. x., p. 57.

474. Morton, A. S. Fibro-myxoma of the plica semilunaris and lower conjunctival sac. *Ibid.*, p. 64.

475. Lopez, Enrique. A clinical note on ophthalmia neonatorum. *Revista de Ciencias Medicas*, Dec. 5, 1890.

Andrews (470) found the gonococcus 17 times in 17 cases of acute gonorrhœa, 108 times in 144 cases of chronic gonorrhœa, and in every case of 72 instances of purulent ophthalmia in adults and in 122 of the newly-born. He thinks there is often a purulent ophthalmia in children under three weeks which is not specific. His main reliance in treatment is in an instillation of a 2-per-cent. solution of nitrate of silver, once or oftener a day, according to indications ; the eye to be kept clean with frequent washings of a boric-acid solution, and cold to be constantly applied. In a total of 153 cases treated, there was a total loss of 1 eye in 8 cases.

BURNETT.

According to SCHMIDT's (471) report, the chief contingent of eye diseases is formed of cases of trachoma, in which he includes the so-called folliculosis and the catarrhus follicularis. He has often observed the development of undoubted trachoma from the lighter forms of folliculosis. In its early stages, trachoma yields readily to treatment. After being cured, the patient should still be kept under observation for a week or two, to guard against relapses. The formation of follicles on the tarsal surface is always a sign of the long duration of the disease, and demands a more extended treatment. In the first stages of trachoma the follicles are present only in the cul-de-sac, and may easily be overlooked. The only cause is infection. The treatment consists in the early squeezing out of the follicles, which usually prevents extensive cicatrization ; and later in the use of 1-per-cent. to 2-per cent. nitrate of silver, 2 per cent. acetate of lead, or the stick of sulphate of copper, which prevents the involvement of the cornea.

HIRSCHMANN.

DARIER (472) operates on the severer cases of granular conjunctivitis after Sattler's manner. He, however, lays less weight on the scraping than on the brushing and washing of the cul-de-sac with sublimate solution 1 : 500.

MARCKWORT.

REID (473) contributes an interesting microscopical study of conjunctival inflammations, illustrated with microphotographs. Lymphoid infiltration is shown to be a frequent condition in various forms of conjunctivitis. In recent follicular conjunctivitis, the follicles have a finely reticulated stroma, are not surrounded with any definite capsule, and probably increase in size by the addition of surrounding lymphoid cells ; as the follicle develops the connective tissue in the centre increases, and fatty or other degeneration occurs. Probably the formation of follicles is secondary to the general lymphoid infiltration. A follicle may burst and discharge on the surface. In cases of long standing goblet cells become numerous, and some may become involuted and thus be the beginning of a mucous follicle forming a vesicle in the retrotarsal fold. The fibro-cellular infiltration is not limited to genuine trachoma, but may occur in chronic follicular cases, so that the distinction between these two diseases is merely clinical. Werner.

Morton (474) gives an illustration of two small fibro-myxomatous tumors *in situ ;* they were firm and pinkish in color, and were growing for three or four months. Patient aged 37. Werner.

Lopez (475), after a twin delivery, found purulent ophthalmia on the third day in the last-born infant, which was delivered with the forceps, while the first-born infant, which was delivered easily, did not contract the disease. This proves (1) that children born of a leucorrhœic mother do not necessarily get purulent ophthalmia ; and (2) that the infection takes place through the direct contact of the secretion with the conjunctiva, for the case could be explained on no other grounds.

476. Feilchenfeld. The therapy of superficial corneal infiltration. *Zehender's klin. Monatsbl. f. Augenheilk.*, vol. xxviii., p. 502.

477. Boullet. Étude comparative du traitement des abscès de la cornée par la chaleur et la froid. *Thèse de Lyon*, 1890.

478. Randolph, R. S. Mild solutions of corrosive sublimate in the treatment of keratitis. *Johns Hopkins Hosp. Bull.*, vol. i., 4, p. 46.

479. Cant, W. E. Case of keratitis from paralysis of left fifth nerve ; injury to fifth nerve within cranium. *Trans. Ophth. Soc.*, vol. x., p. 233.

480. Despagnet. Traitement du keratocone pellucide. *Soc. d'Opht. de Paris*, Nov. 4, 1890.

Feilchenfeld (476) scratches out superficial corneal ulcers with a gouge or needle, and then applies an antiseptic dressing. The results are good.

Randolph (478) has found instillation of solution of the mercuric chloride, from $\frac{1}{6000}$ to $\frac{1}{12,000}$, every four hours useful in the treatment of all forms of keratitis. Atropine solution is used in addition. Burnett.

Cant's (479) patient had complete paralysis of all motor and sensory branches of the fifth nerve. The boy was aged sixteen, and had received a blow from a reaping-hook two or three weeks before, the point of the hook having penetrated the squamous portion of the temporal bone to the depth of an inch. Severe keratitis set in, which improved after slight return of sensibility.

WERNER.

DESPAGNET (480) reported three cases of keratoconus treated in different manners. In the first case a small ulcer formed at the apex of the cone. A compressed bandage was used and later, after cauterization with nitrate of silver, the cone became flatter, but a slight keratoglobus resulted. In the second case, one eye was treated with eserine and a compress bandage, while the other eye was not treated. The conus increased equally in both eyes and the vision decreased correspondingly. In the third case he excised a crescentic bit from the entire thickness of the cornea after Galezowski's method. The wound cicatrized in a month, but six months later became infiltrated and an ulcer developed. This was cauterized with the galvano-cautery, a paracentesis was made and a compress bandage applied, and the ulcer healed. Despagnet, therefore, recommends the treatment by excision of a crescentic bit of cornea. In the discussion de Wecker advised against operative treatment. Of all the operations suggested that of v. Graefe is still the best. Meyer operates according to v. Graefe. Abadie perforates the cornea with the galvano-cautery. Jocqs favors eserine and a compress bandage. Gillet de Grandmont saw a good result after v. Graefe's operation, but will not hesitate to open the anterior chamber in the excision. Parinaud scarifies the cornea.

MARCKWORT.

481. MILLIKEN, B. T. A case of foreign body remaining in the eye for twenty years, followed by abscess in the scleral wall. *Med. News*, Nov. 8, 1890.

MILLIKEN (481) relates an interesting case of a piece of cap remaining in the eye, with fair vision for twenty years. It then gave rise to disturbance which was repeated at intervals for ten more years. The foreign body had entered at the edge of the cornea and passed through the iris and lens, which latter was circumscribedly opaque. With the ophthalmoscope the body could be seen projecting into the vitreous. It later formed a projection on the outer surface of the sclera and finally M. punctured the circumscribed tumor and let out three or four drops of pus and some metallic detritus. The patient has now $V = \frac{3}{30}$ and all symptoms of irritation have disappeared.

BURNETT.

Sections XII.–XXI. Reviewed by Dr. P. SILEX.

XII.—IRIS.

482. Brailey. Double microphthalmos with defective development of iris, teeth, and anus. Glaucoma at an early age. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 139.

483. Hill, Griffith. Case of tuberculosis of the iris. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 84.

Brailey's (483) patient, a girl æt. 18, had only a crescentic portion of iris left at inner side in the right eye, and three small, isolated bits in the left eye. The corneæ measured 9 *mm* in diameter. Myopia = 12 D. Vision only = counting fingers. Disks cupped. Werner.

In Griffith's (483) patient, a child æt. 7 months, a yellowish non-vascular tumor grew at ciliary border of the iris at outer side, pupil closed by delicate membrane and there was slight bulging of sclero-corneal region. No history or signs of phthisis or syphilis. On account of further formation of nodules the eye was enucleated. The child died nine months after from tubercular meningitis. The growth exhibited a tubercular structure, but bacilli could not be demonstrated probably owing to hardening fluid having been bichromate of potash. The paper concludes with an interesting review of previously recorded cases and a list of references.

Werner.

XIII.—CHOROID.

484. Brailey. Cyclitis, probably of gouty origin. *Trans. Soc. of United Kingd.*, vol. x., p. 96.

Brailey's (484) patient, a man æt. 36, after having had six attacks of gout in the feet and limbs, became suddenly affected with well-marked cyclitis in one eye which relapsed three times. The eye was acutely tender to the touch and periphery of anterior chamber deep. Werner.

XIV.—GLAUCOMA.

485. Collins, J. T. Glaucoma after extraction of cataract. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 108.

486. Morton, Stanford. Hemorrhages on the disk in a case of glaucoma (with plate). *Ibid.*, vol. x., p. 153.

487. Smith, Priestley. On the size of the cornea in relation to age, sex, refraction, and primary glaucoma. *Ibid.*, vol. x., p. 68.

488. KEYSER, P. A case of glaucoma fulminans of peculiar interest. *Am. Jour. of Ophth.*, No. 8, 1890.

In this interesting paper COLLINS (485) describes the history and pathological condition of 10 eyes which were lost from glaucoma after cataract operations. In 9 cases the cataract was senile, in one probably traumatic. In 5, one eye was operated on without any subsequent symptoms of glaucoma, in 3 the increased tension came on after needling, and in 3 others it was accompanied by iritis with keratitis punctata, and sympathetic ophthalmia occurred.

The glaucoma came on in from 3 months to nearly 2 years after the operation. Iridectomy was performed in 9 cases of extraction. Pathological examination revealed adhesion of lens capsule to the wound except in one instance where the hyaloid of the vitreous was adherent to it, and in this case the lens had been removed in its capsule, and in all cases except this some cortical remains had been left in the peripheral portion of the capsule. The angle of the anterior chamber in the coloboma was blocked in all cases, and elsewhere there was peripheral adhesion of the root of the iris except in the kerato-iritic cases where there was cellular infiltration of lig., pectinatum iridis, etc.

From the beginning of 1885 to the middle of 1889 1,405 senile cataracts were extracted at the Moorfields Hospital and 9 of these were lost from glaucoma, or 0.64 per cent. WERNER.

In MORTON'S (486) case the hemorrhages on the margins of the cupped disk were associated with aneurismal dilatations of some vessels. WERNER.

P. SMITH'S (487) investigations were undertaken to test the truth of a previous suggestion of his, that small corneas were associated with a liability to primary glaucoma. 1,000 normal eyes were examined; ages of subjects 5 to 20. Average horizontal diameter 11.6 *mm*, the maximum being 13.5 *mm*, and the smallest 10.5 *mm*. The cornea attains its diameter very early in life—about the age of 5. The relation which sex and refraction have to the diameter of the cornea is almost negative. 178 glaucomatous eyes examined gave an average diameter of 11.12 *mm*, maximum 12.5, minimum 10 *mm*. Corneas with less than 11 *mm* diameter formed 25 per cent. of glaucomatous and less than 4 per cent. of normal eyes. The smallness of the cornea is not a consequence of the glaucoma, as patients who had one eye normal had the same-sized corneas in both eyes. Two questions still remain to be positively determined—namely, are small, corneas associated with a proportionally small lens and eyeball. WERNER.

In KEYSER's (488) case after an extraction with a large iridectomy there remained a thickened capsule which was finally drawn out through a small corneal incision by means of a Tyrrel hook. After this latter operation there developed in a few hours an attack of glaucoma fulminans, which was subdued by active treatment, but recurred twice subsequently. The final result however, was good. BURNETT.

XV.—SYMPATHETIC OPHTHALMIA.

489. GALEZOWSKI. Du mode de transmission de l'ophtalmie sympathique et de son traitement. *Soc. d'Opht. de Paris*, Oct. 7, 1890.

490. GOODE, G. A. A case of sympathetic ophthalmia two weeks after enucleation of the injured eye. *Jour. Am. Med. Asso.*, July, 1890.

491. MEYER. Quelques remarques sur l'ophtalmie sympathique. *Soc. d'Opht. de Paris*, Nov. 4, and *Rev. génér. d'opht.*, 1890, No. 11., p. 481.

492. DE WECKER. Resection simple du nerf optique *Soc. d'Opht. de Paris*, Nov. 4, 1890.

493. ROLLAND. Traitement préventif de l'ophtalmie sympathique. 1890, p. 527.

GALEZOWSKI (489) assumes that sympathetic inflammation is conveyed from one eye to the other through the ciliary nerves and their lymph channels, and not through the optic nerves. He bases his opinion on the anatomical investigations of Poncet in opposition to Knies, Leber, and Deutschmann. MARCKWORT.

GOODE's (490) patient was a boy of eleven years whose cornea was ruptured by a blow from a stone. Two weeks after the eye was enucleated, and fourteen days later a sero-plastic iritis set up in the fellow-eye. Vision at last report only $\frac{20}{60}$. BURNETT.

MEYER (491) in a long paper discusses Deutschmann's experiments on ophthalmia migrans in their relation to clinical observations. Meyer grants the value of these investigations in part, but believes that sympathetic ophthalmia in man does not arise from the simple migration of bacteria to the second eye through the optic nerve. He advises early enucleation. In glaucoma absolutum Meyer has made æquatorial punctures for several years.

MARCKWORT.

de Wecker (492) advises resection of the optic nerve in place of enucleation for sympathetic ophthalmia. His operation differs from those of Schweigger and H. Pagenstecher, in that he endeavors to limit it as much as possible to the nerve itself. He draws the eyeball forward with his "crochet articulé," and seizes the tense nerve with his "ciseaux compresseurs," and excises as large a piece of the nerve as possible. Marckwort.

Rolland (493) believes enucleation to be the only safe and commendable operation for sympathetic trouble. He warns practising physicians not to be misled by de Wecker's paper into the belief that enucleation can be substituted by de Wecker's æquatorial punctures. de Wecker's paper is severely criticised.

Marckwort.

XVI.—LENS.

494. Berry. A case of spontaneous purulent hyalitis occurring nine months after a successful cataract extraction, with remarks. *Trans. Ophth. Soc. of the United Kingd.*, vol. x., p. 160.

495. Collins, E. T. Lens with epithelium on its posterior capsule and other peculiar structural changes. *Ibid.*, vol. x., p. 165.

496. Tyner, J. D. Preliminary capsulotomy in the extraction of cataract. *New York Med. Jour.*, Sept. 30, 1890.

497. Lippincott. On intra-ocular syringing in cataract extraction, with a report of fifty-three operations. *Amer. Jour. of Ophth.*, July, 1890.

498. Michel, M. History of the admission of light in the after-treatment of cataract operations. *Med. News*, Sept. 20, 1890.

499. Snell, S. On artificial ripening of cataracts with special reference to Förster's operation of trituration. *Trans. Ophth. Soc. of the United Kingd.*, vol. x., p. 214.

500. Thompson, J. T. Note on a case of hereditary tendency to cataract in early childhood. *Ibid.*, vol. x., p. 141.

501. Webster, D. Traumatic dislocation of the crystalline lens with increased tension and severe pain; relief of pain and restoration of useful vision follow extraction. *New York Med. Jour.*, Sept. 13, 1890.

In Berry's (494) case the patient, a glass-blower, returned to his occupation a month after a successful cataract extraction; nine months later he presented himself with a purulent hyalitis, with hypopyon. The eye was enucleated. No adhesion of iris to the wound, which was firmly united. The suppuration seemed to have begun just behind the corneal margin. A second similar case is recorded in the paper. The patient was a cook. WERNER.

COLLINS' (495) patient lost the eye from secondary glaucoma due to a choroidal growth. The lens exhibited a single layer of nucleated epithelial cells lining the posterior capsule, and a central band of hyaline bodies. WERNER.

TYNER (496), after dilating the pupil, introduces a Bowman needle and ruptures the lens capsule in its upper quadrant. The incision is then made and the extraction performed in the customary manner. The aqueous does not escape. BURNETT.

LIPPINCOTT (497) tabulates fifty-three extractions, in all of which a preliminary iridectomy had been made in which he washed out the corticalis with a boric-acid solution by means of a specially devised syringe. His successes were ninety-eight per cent. ; failures, two per cent. BURNETT.

MICHEL (498) shows that the reaction against dark rooms and heavy bandages began as far back as 1801 with Beer, and that Walton and others for closing the lids used adhesive straps only, in 1870. Among Americans, Agnew of New York and Lewis and Roberts of Philadelphia used the same methods. The method was brought more prominently forward however by Dr. Chas. E. Michel of St. Louis, and has since been followed particularly by Chisolm of Baltimore. BURNETT.

SNELL (499) speaks favorably of Förster's operation. He reports nine cases, only two of which were unsuccessful, and one of these had no cortical capacity. In no case was there iritis or any complication. In the discussion Tweedy stated that he had frequently operated on unripe cataracts with successful result, which he attributed to the peripheral rupture of the capsule. Hill-Griffith had tried trituration in twenty-eight cases on the anterior portion of the capsule, and once a partial dislocation of the lens occurred. Eales had seen no bad result in thirty cases. In a number of cases there was slight adhesion between the iris and the anterior capsule. Vitreous was never lost. Brailey does not favor the extraction of unripe cataracts. WERNER.

THOMPSON (500) gives a genealogical tree of five generations in which eleven persons developed cataract before five years of age ; in two generations this seems to have been transmitted through the male line. The cataracts were not zonular but more like senile in density. WERNER.

WEBSTER's (501) patient was a man of sixty years. In extracting the lens he did not use the trident devised by Dr. Agnew, but after making a large incision introduced a wire spoon and picked the transparent lens out in its capsule. BURNETT.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

502. EDMUNDS, W. Sections of retina from a case of leucocythæmia. *Trans. Ophth. Soc. United Kingd.*, vol. x., p. 157.

503. EPÉRON. Sur quelques symptomes tabiformes de l'amblyopie toxique. *Rev. méd. de la Suisse romande*, Aug. 8, 1890.

504. EWETZKY. On the pathological anatomy of retinitis pigmentosa. *Wjestnik Ophth.*, No. 6, 1890.

505. GARAFOLO. A case of quinine amaurosis. *Wiener med. Blätter*, No. 15, 1890.

506. GALLEMAERTS. Amblyopia from sulphuret of carbon. *Ann. d'ocul.*, vol. civ., p. 154.

507. GRAND, CLÉMENT. Efficacité des injections d'antipyrine dans trois cas d'héméralopie symptomatique de rétinite pigmentaire. *Ann. d'ocul.*, vol. civ., p. 232.

508. HIRSCHBERG. On retinal changes due to old age. *Centralbl. f: prakt. Augenheilk.*, vol. xiv., p. 322.

509. KOENIG. Les thromboses artérielles de la rétine. *Rec. d'opht.*, 1890, p. 697.

510. LAWFORD, B. Embolism of a branch of the arteria centralis retinæ. Right eye. *Trans. Ophth. Soc. United Kingd.*, vol. x., p. 153.

511. WERNER. Abnormal retinal veins. *Ibid.*, vol. x., p. 151.

512. FERNANDEZ. Treatment of detachment of the retina by scleral puncture. *Anales de la Real Academia de Ciènces med. fis. y natur*, Dec., 1890.

In EDMUNDS' (502) case, in addition to hemorrhages, the retina showed thickening of the walls of the arteries, and clots of white corpuscles in the veins. WERNER.

EPÉRON (503), in cases of intoxication amblyopia, has frequently seen tabic symptoms, such as the Argyll-Robertson pupil, myosis, loss of knee-jerk, Westphal's symptom, etc. The chief differential points are the preservation of the periphery of the visual field and the existence of a central scotoma. MARCKWORT.

In examining the eyes of a man of forty-four, dead of delirium tremens, who had been blind from early youth, EWETZKY (504) found the retinas moderately pigmented, particularly between the equator and the disk.

Microscopically the inner layers of the retina were seen to be well preserved. The outer molecular layer, the outer granular layer, and the rods and cones were entirely wanting. They were replaced by a thin layer of reticular connective tissue lying on the inner granular layer. In the nerve-fibre layer the nerve fibres were thickened at many points, as in retinitis albuminurica. The vessel walls were thickened, the lumen narrowed and at some points obliterated, and many vessels were transformed into connective tissue. Pigment in the form of free-lying granules was present in all the layers of the retina, but particularly in the nerve-fibre layer, and less chiefly about the vessels. The pigment epithelium was altered, the form of the cell remaining usually normal, but the pigment altogether wanting or much decreased, and often accumulated between the cells.

The author concludes from his studies that retinitis pigmentosa is a disease of the retinal vessels, which leads to atrophy of the outer layers of the retina and secondary hypertrophy of the connective tissue. The pigment is washed out of the atrophic pigment epithelium by the fluid currents in the inner layers of the retina. HIRSCHMANN.

GARAFOLO'S (505) patient, a man, æt. sixty-one, took 15 grm. of quinine. Six days later R V = $\frac{6}{6}$, L V = $\frac{6}{36}$, with marked contraction of the field of vision, and decrease in the light sense of the entire retina. The disk was pale and the vessels narrow.

GALLEMAERTS (506) describes a case of amblyopia from sulphuret of carbon poisoning. The patient was 18½ years old, and his occupation was dipping caoutchouc in sulphuret of carbon in a factory. There had been no abuse of tobacco or alcohol. Besides the amblyopia there were other symptoms, such as muscular weakness, cramps in the calves of the legs, arms, and abdomen, and poor hearing. Central scotoma for all colors, and V = fingers at 45 *cm*, both. After the patient gave up his occupation the vision improved in two months to R = $\frac{1}{20}$, L = $\frac{1}{4}$.

MARCKWORT.

HIRSCHBERG (508) in 98 eyes of individuals between sixty and eighty found but 22 normal. For the most part there were slight changes in the retina, attributable to arterial disease.

Koenig (509) described three old and one new case of thrombosis of the retinal arteries. Ophthalmoscopically the vessels affected most frequently show the blood current interrupted by white masses at intervals. At times the coagulæ are not white, but yellow. The vessels may also be entirely obliterated.

Marckwort.

Lawford's (510) patient, a man æt. seventy-one, with dilated heart, partially lost his sight from embolism of one of the branches of the lower division of the central artery, the position of the embolus being visible. Werner.

Werner (511) gives an illustration of a horse-shoe-shaped vein dipping into the disk at both its extremities. Werner.

Fernandez (512) describes a case of traumatic detachment of the retina downward and outward of six weeks' standing, cured by puncture of the ocular membranes 5 *mm* behind the cornea. The puncture gave exit to a clear fluid. Two months later a bluish white patch, due to partial atrophy of the choroid, could be seen with the ophthalmoscope at the point where the detachment had been. The author attributes this exceptional result to the fact that the detachment was traumatic, the eye being previously healthy.

XIX.—OPTIC NERVE.

513. Hansen, M. A case of acute retrobulbar neuritis. *Med. Record*, Nov. 8, 1890.

Hansen reports a case of a woman who, after an attack of influenza, was taken with severe headache, which gradually increased in violence. There was slight fever, but no delirium or unconsciousness. After some days V began to fail, and in three days was entirely gone. Pupils dilated ad maximum, and did not respond to light. The ophthalmoscope showed signs very like embolism of the central artery of the retina ; veins large, arteries very small, and disks whitish. No heart disease, and urine normal. The retinal arteries gradually filled up, and vision returned to $\frac{5}{200}$, at which it remained stationary. The disk still appears whitish. Burnett.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

514. Doyne, R. Perforation of cornea and lens by a chip of steel without subsequent opacity of the lens. *Trans. Ophth. Soc. Unit. Kingd.*, vol x., p. 195.

515. Gazis. Çorps étranger enclavé entre l'iris et la cornée. *Rec. d'opht.*, 1890, p. 638.

516. Gunn, M. Persistent mydriasis following comparatively slight injury. Corresponding cicatrices in cornea and anterior capsule. Advanced globular degeneration of lens. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 196.

517. Huysmann, A. Paralysis n. laryngei (securrentis inf.) completa et ptosis sympatico-paralytica (Horneri) traumatica. *Weekblad van het Nederlandsch Tydschrift voor Geneeskunde*, 1890, ii., p. 790.

518. Schleich. A case of subretinal cysticercus, with remarks on the frequency of cysticercus in Wurtemberg. *Med. Correspondenzblatt des Würtemberg. ärztl. Landes-Vereins*, vol. lx., No. 22, p. 169.

519. Snell, S. The employment of the electro-magnet in ophthalmic surgery. Additional cases and remarks. *Brit. Med. Jour.*, Nov., 1890.

Gazis (515) reports a case in which a piece of iron, 4 *mm* long and 2 *mm* wide, flew into the eye and remained fixed between the cornea and the iris. An attempt at extraction with forceps was unsuccessful. Even after an iridectomy the foreign body could not be removed with forceps, but when an electro-magnet was introduced the iron was easily extracted. A short time previously the author extracted a piece of iron from the anterior chamber in a similar manner. The foreign body was encapsuled in an exudation, was not visible, and its presence was doubted. When the magnet was brought near the eye the patient felt a movement, and the exudation surrounding the foreign body moved freely, following the motion of the magnet outside the cornea. The diagnosis being thus made, the anterior chamber was opened and the bit of iron easily removed. Marckwort.

Gunn (516) observed a case of permanent mydriasis after a comparatively trivial injury. There were cicatrices in the cornea and lens corresponding in position. A number of myelin globules were seen in the anterior capsule of the lens. There was no rupture of the sphincter iridis, since eserine caused an energetic reaction. Werner.

Huysmann's (517) patient was wounded in the neck with a small knife. The wound healed rapidly, but soon a peculiar hoarseness was observed, with slight ptosis of the right eyelid and a red color in the right cheek. The right pupil was round, but smaller than the left, and reacted more sluggishly. No further changes. The hoarseness depended upon a total immobility of the right half of the larynx. The sensibility of the mucous membrane was not altered. In the neck there was a cicatrix 18 *mm* long. Huysmann's diagnosis was: Laesio n. vagi dextr. et n. sympathici cervicalis (gangl. medii ?).

SCHLEICH (518) found that in twenty years only four cases of intraocular and two of subconjunctival cysticercus had been observed in Wurtemberg. Tænia medio-cannellata is seen more frequently than T. solium.

SNELL (519) reports thirty-seven new cases in which he has employed the electro-magnet. In nine cases the foreign body was in the anterior chamber, in twelve cases in the vitreous. Of the rest one was in the tear sac and the others in the conjunctiva, cornea, and sclera. Cases in which the foreign body lies in the anterior chamber are the most favorable for restoration of vision, those on the contrary in which the foreign body is in the vitreous are the most unfavorable. WERNER.

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

520. ABERCROMBIE, J., and GUNN. A case of uniocular proptosis with double papillitis and intracranial bruit. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 243.

521. ANDERSON, J. Homonymous hemianopsia; recovery; subsequent death and necropsy. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 243.

522. BRAUNSTEIN. Ocular affections with influenza. *Wjestnik Ophth.*, Nov.–Dec., 1890.

523. DOYNE, R. Recovery from hemianopsia; subsequent necropsy. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 235.

524. GAZIS. Des complications oculaires à la suite de l'influenza. *Red. d'opht.*, 1890, p. 586.

525. HENSCHEN, S. Clinical and anatomical contributions to the pathology of the brain. Part I., with 36 plates and 3 diagrams. Upsala, 1890.

526. TILLEY, R. Report of a case of homonymous hemianopsia with a wound in the occipito-parietal region. *Jour. of the Amer. Med. Assoc.*, Oct. 11, 1890.

527. HARDIE and C. A. WOOD. Two cases of nasal hydror-

rhœa with a report on the eye-symptoms. *New York Med. Jour.*, Sept. 6, 1890.

528. KOENIG. De l'artério sclerose et des affections oculaires qui en dépendent. *Thèse de Paris*, 1890.

529. LIEBREICH. Remarkable cases of Basedow's disease, observed in Prof. Schöler's clinic. *Zehender's klin. Monatsbl. f. Augenheilk.*, 1890, p. 492.

530. MORTON, A. S. Alexia with right-sided hemianopsia. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 247.

531. NOYES, H., and DANA, L. A case of tumors of the ventricle with left anophthalmus. *Med. Record*, July 16, 1890.

532. PANAS. Rapport de l'ophtalmologia avec la médecine générale. *Rec. d'opht.*, 1890, p. 633.

533. DE SCHWEINITZ, G. The ocular complications of malaria. *Med. News*, June 7, 1890.

ABERCROMBIE and GUNN (520) describe the case of a child, æt. two and one-half years, who had an intracranial bruit, heard all over the head ; there was double optic neuritis with proptosis of left eye. Some history of a headache ; no injury. In six months the child was almost blind, disks white. Proptosis unaltered. WERNER.

In ANDERSON'S (521) case, a man, æt. forty-one, there was failure of vision with severe frontal headache, loss of flesh. He was found to have right hemianopsia, the fields varied a little from time to time, he subsequently had vomiting, and a transitory attack of left hemiplegia. *Post mortem* a glioma of the left angular gyrus was discovered. Nothing to account for the hemiplegia. WERNER.

BRAUNSTEIN (522) reports the following cases : I. A girl, æt. fourteen, previously strong and healthy, had an attack of typical epidemic influenza with high temperature, severe headache, and vomiting. Soon after her recovery she noticed diplopia, strabismus and giddiness. A month after the influenza the tendon reflexes in the upper extremities were weak, the patellar reflex altogether wanting. Sensibility of skin of right leg diminished. Pupils dilated, and reacting sluggishly. Bilateral abducens paralysis (right more marked). Severe neuro-retinitis, both. V = $\frac{20}{100}$ O. U. Diagnosis : neuritis multiplex. Treatment : hot foot-baths, cantharides to the neck, internally infus. jaborandi with sod. salicyl. Rapid marked improvement. II. A healthy man had an attack of typical influenza and noticed on the fifth day, when the temperature was lower, that his vision was poor, and twelve hours later he was entirely blind. Examination some days later showed complete blindness in both eyes, disks of normal color, perhaps somewhat hazy, and the retina slightly hazy. Arteries small, veins tortuous and dilated. Temperature normal. Pulse 130; otherwise normal condition. No results from treatment. Diagnosis: probably retrobulbar neuritis (ischæmia retinæ, in consequence of weakness of the heart not excluded). III. In a tabic patient, æt. fifty-three, a serous iritis developed in the left eye, one week after an attack of influenza. It ran its natural course. IV. A severe suppurative choroiditis in one eye in the course of a typical influenza. HIRSCHMANN.

DOYNE'S (523) patient, a man, æt. seventy, suddenly became confused and was found to have right hemianopsia, the vision and fields altered from time to time, the right halves recovering, and the left lower quarters were at one time lost. He subsequently developed paresis of the left arm and right side of face, and aphasia. He died unconscious. Left hemisphere of brain, small patch of softening at upper end of perpendicular fissure. Right cerebral hemisphere, three patches of softening, one cortical near tip of occipital lobe, the two others also in occipital lobe.

WERNER.

GAZIS (524) describes the complications of the influenza that were observed in Galezowski's clinic. They were as follows: Choked disk, herpes corneæ, conjunctivitis, and episcleritis. Gazis speaks in detail of the choked disk and reports a new case of that sort. MARCKWORT.

HENSCHEN'S (525) work, which is of great value respecting the course of the visual tracts in the brain and the clinical symptoms of hemiopia, cannot satisfactorily be reviewed here on account of its size. The microscopic illustrations were prepared from not less than ten thousand preparations. A detailed review may be found in the *Berliner klin. Wochenschr.*, 1890, p. 1096.

TILLEY (526) reports the case of a young man with a typical left homonymous hemianopsia which seemed to follow a blow on the back part of the head. The pupils contracted when light was thrown on the blind part of the retina. Syphilis seemed to be excluded, and otherwise the patient appeared in good health.

BURNETT.

The eye symptoms noted by HARDIE and WOOD (527) in the cases of nasal hydrorrhœa were dimness of vision and a concentric narrowing of the visual fields. There was no color-blindness. The disks showed strips of atrophy at the periphery of the nerve entrance. Fundus otherwise normal. BURNETT.

As complications of Basedow's disease, LIEBREICH (529) observed an ulceration of the cornea in both eyes, a constant peculiar twitching of the lids, twice an ophthalmoplegia externa, and once a paresis of the right abducens.

In the case of NOYES and DANA (531) the man had lost the left eye by enucleation in 1873, fifteen years before he was seen by them. Besides the other general symptoms of brain tumor, those referable to the eyes, were : paralysis of sixth nerve on the right side, branches of the third to the levator palp., the sixth on the left side, irritation of fourth and the branches to sup. and inf. recti, causing rotating and vertical nystagmus. A section of the decussation of the optic nerves showed a band of degenerating tissue crossing from the upper part of the left nerve to the lower part of the right tract ; there was no degenerated band in the left tract, but there was in the right, seeming to show a total decussation of the nerve fibres. The eye symptoms also seem to indicate a larger nucleus for the levator palp. than has been supposed to exist. The ciliary muscle and iris were not involved. BURNETT.

PANAS (532) reports the case of a boy, æt. seven, in whose right eye there had been severe hemorrhage into the vitreous ; in the left there was seen the disease which Manz had described as retinitis proliferans. There was an organized plaque in the external portion of the disk and in front of this opacities in the vitreous. These plaques were of hemorrhagic origin. The ocular hemorrhages, according to Panas, were induced by a vaso-motor reflex proceeding from the intestinal canal. The patient suffered with recurring diarrhœa. MARCKWORT.

DE SCHWEINITZ (533) gives the histories of several cases, and cites the literature of the whole subject of the ocular complications of malaria, and concludes : (1) that there is an intermittent ophthalmia, malarial in its nature ; (2) that there is a true and distinct malarial keratitis ; (3) that various functional disturbances, such as amblyopia, paresis of accommodation, alterations in the visual field, hemianopsia, and night blindness, can be malarial in origin ; and (4) that there may be gross changes from the same cause, such as neuritis, atrophy of the nerve, and hemorrhages. BURNETT.

REPORT ON THE OPHTHALMOLOGICAL SESSION OF THE SPANISH MEDICAL CONGRESS, HELD IN VALENCIA, JULY, 1891.

Treatment of Granulations by Means of the "brossage."—Dr. Osio, of Madrid, dwelt on the tenacity of this disease and the inefficiency of almost every treatment, some of them being even dangerous, such as the employment of *jequirity* and excision of the conjunctival cul-de-sac. He generally resorts to the "*brossage*" with a hard tooth-brush, after having inverted the eyelids and scarified the conjunctiva. He performs the inversion of the eyelids with a forceps especially devised for this purpose. The brushing, when performed after scarification, has given a most prompt success.

Dr. Lloret, of Valencia, is of the same opinion, and pronounces himself against the excision of the cul-de-sac suggested by Galezowski, which he believes is apt to produce entropion by cicatricial retraction.

Dr. Santos Fernandez, of Havana, said that all the methods of treatment suggested against granulations are deficient because they are unphysiological; they succeeded in removing granulations by destruction of the conjunctival epithelium, this being the ultimate result of granulations when left alone. He agrees that the excision of the cul-de-sac is a defective method, but affirms that it is free of danger; he has considerable experience in it. Knapp's treatment by pressing out the trachomatous substance with special roller forceps is the only conservative treatment, but he has not yet formed an opinion as to its efficiency.[1] In the meantime he accepts brossage, notwithstanding the necessary destruction of epithelium, for this method is the only one that will certainly extinguish granulations.

Dr. Aguilar Blanch believes that "excision" is free of danger, and that there is no single treatment that can be resorted to in every case ; the treatment must be different, according to the nature of the disease.

---

[1] I have operated on fifty-six cases. The results are most satisfactory. No scars where there were none before. Treatment in one sitting. No relapses. —Sept. 8, 1891.—H. Knapp.

Loss of Sight in Yellow-Fever.—Dr. SANTOS FERNANDEZ read a paper on this most rare complication ; he had only heard of three cases ; one he had observed himself, and the other two he had been informed of by two colleagues. Ophthalmoscopic examination was performed in two cases, and it was proved that the eye was free from organic lesions. In the case which he observed himself, anuria and violent delirium were present ; all three patients died within the first week.

Dr. OSIO, by comparison with other infectious diseases, thinks that the loss of sight is due to anuria.

Ocular Lepra.—Dr. AGUILAR BLANCH : In ocular lepra the eyelids are affected with anæsthesia, nodules, naderosis, and ectropion ; entropion is of rare occurrence ; the conjunctiva is also involved in the same processes ; the cornea is also inflamed late ; the sclerotic and iris are not spared ; the iris is generally subject to chronic inflammation.

Dr. SANTOS FERNANDEZ agrees with the reader, for he has been able to confirm every fact in the Havana Leper Hospital. The same is the case with his colleague, Dr. Lopez, author of the paper mentioned by Dr. Aguilar ; in a doubtful case diagnosis was established by bacteriological examination.

New Method for the Correction of Strabismus.—Dr. AGUILAR isolates the muscle and the capsule from the sclerotic ; he does not divide the muscle nor does he employ any suture ; the tendon is afterwards shortened by suture. He exhibited a new instrument for the measurement of the degree of retraction.

Dr. SANTOS FERNANDEZ says that if the method would not have proved successful in the hands of the author he would for theoretical reasons have deemed it inefficient.

Dr. OSIO thinks that instruments for the measurement of the muscular retraction are poor in practical results.

Inheritance of Cataract.—Dr. AGUILAR speaks of a family whose members are strongly predisposed to cataracts, and shows the cases arranged in a table.

Dr. SANTOS FERNANDEZ has observed hereditary cataracts in a branch of his own family, and thinks that in the explanation of these facts a place must be left for mere coincidence. Thousands of cases are treated that show no family predisposition.

Post-Traumatic Cataract.—Dr. VIRCIANO reports a number of these cases ; he endeavors to explain the fact by the opacity of the crystalline lens, and asks whether these cataracts should be operated on or not.

Dr. SANTOS FERNANDEZ says that traumatisms are sometimes overlooked by the patients; that cataracts are more satisfactorily treated in old than in the young persons.

Method for the Removal of Staphyloma.—Dr. OSIO simply cuts off the staphyloma, passing his knife before the iris, and finishes the removal of the opaque cornea with the scissors; after this he applies a compressive bandage; the presence of the iris largely contributes to the normal appearace of the eye.

Treatment of Short-Sightedness by Extraction of the Crystalline Lens.—Dr. SANTOS FERNANDEZ has observed the visual power increased in myopes after the operation for cataracts; he consequently thinks this method can be resorted to in those cases in which short-sightedness has so much progressed as to constitute a serious danger for the retina. He has recently seen two successful cases in Dr. Abadie's ophthalmological clinic. He thinks that discission should be performed in the young and extraction in the old.

Dr. AGUILAR agrees with Dr. Santos Fernandez, but thinks that before transparent crystalline lenses are to be extracted more evidence is needed.

Dr. BAYARRI relates a case which strongly confirms the opinions of Dr. Santos Fernandez.

Dr. OSIO does not think that myopes should be treated by extraction, because this would expose them to a useless danger.

Dr. SANTOS FERNANDEZ says that he only proposes the method for those cases over 20 dioptries, as the length of the anterior diameter is shortened by the operation, thus lessening the dangers of the most advanced degrees of progressive myopia.

Ocular Auto-Infection.—Dr. SANTOS FERNANDEZ reported two cases in which suppurative irido-cyclitis had taken place several days after the operation. In one case the infection was attributed to a focus of suppuration of the face, probably due to the irritative action of bichloride; in the second case, as no cause was found responsible for the infection, it was deemed endogenous, and probably due to the weak resisting power of the patient.

Dr. AGUILAR admits auto-infection only in those cases where no filtering surface is left.

Dr. CANDELA said that he admits auto-infection, although the cases are not always clearly understood, because he has observed and thoroughly studied them in obstetrics.

CORRIGENDA.

I.—In Dr. A. Percival's paper on Prismospheres, in this volume, the following errors should be corrected.

P. 195, line 6 from bottom, *for* "the focal distance of the lens; D represents," *read* "the focal distance of the lens in millimetres; if D represents."

P. 199, line 14, *for* "divergence is required, the decentration must be inwards," *read* "convergence must be maintained, the decentration must be outwards."

P. 199, line 19, *for* "outwards $2 \chi 2.661$ *mm*," *read* "inwards 2×2.661 *mm*.

P. 199, footnote, *for* "$10 \tan 7^\circ = .174551 = \tan 9^\circ - 54'.0767$," *read* "$10 \tan 1^\circ = .174551 = \tan 9^\circ\ 54'.0767$."

P. 200, line 16, *for* "object," *read* "defect."

P. 202, after the first paragraph insert the fourth paragraph on p. 203. After the second paragraph insert the third paragraph on p. 203.

P. 203, line 5 from bottom, add after "prismospheres," "in suitable cases."

P. 204, column 1 D, 4th line *for* "31.120" *read* "31.121."

P. 205, line 5 from bottom, *for* "presented," *read* "presumed."

P. 205, last line, *for* "junction," *read* "function."

P. 206, column — 4 D, line 8, *for* "— 2.6," *read* "2.6."

P. 206, column — 7 D, line 3, *for* "26.6," *read* "26.7."

P. 206, last line, *for* "junction," *read* "function."

II.—In my paper "On the Proposed Methods of Numbering Prisms" (ARCHIVES OF OPHTHALMOLOGY, xx., 3), the words "and which has been copied in Prentice's article on 'The Prism-Dioptry' (ARCH. OF OPHTHALM., xix., 2 and 3, p. 128)" should be expunged. The reference should have been to Percival's paper in the same number, p. 257, of the ARCHIVES.

In Prentice's paper, the formula given, namely:

$$R = \sqrt{P^2 + Q^2},$$

in which R, P, and Q represent prism-dioptries and therefore tangents, is practically the same as the amended formula:

$$\text{Tan. } R = \sqrt{\text{Tan.}^2\, H + \text{Tan.}^2\, V},$$

given on page 326 in my article.

A. DUANE, M.D.

ANNOUNCEMENT.

Owing to the steady increase of important contributions (freely illustrated by numerous wood engravings and superb lithographic and colored plates) which have been accepted for publication in the Archives of Otology, the Publishers find themselves compelled to raise the annual subscription price from $3 to $4. The latter is the price at which the German edition, though costing less money to produce, has been issued ever since the separation of the Archives of Ophthalmology and Otology into two independent periodicals. No reduction can be afforded to subscribers taking the two journals, Archives of Ophthalmology, and Archives of Otology, and the subscription for the two will be hereafter $9 a year. The increase in price will go into effect January, 1892.

G. P. PUTNAM'S SONS, Publishers.

September, 1891.

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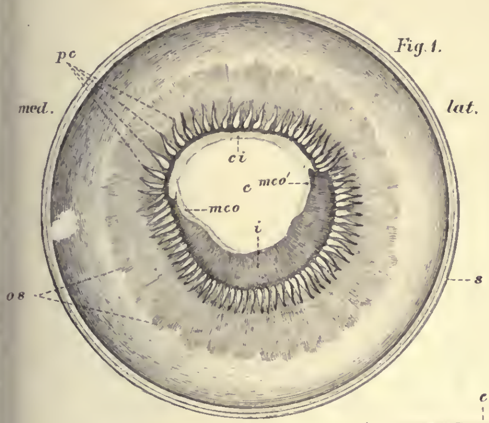

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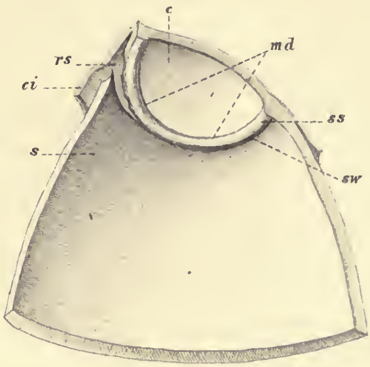

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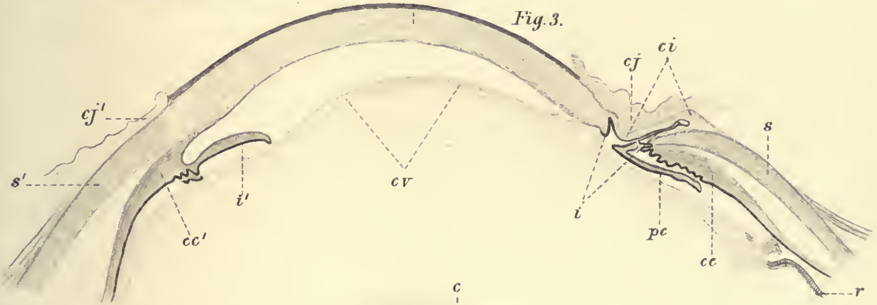

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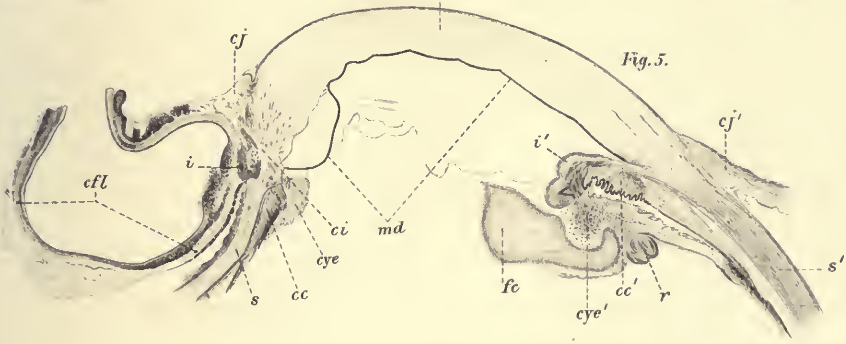

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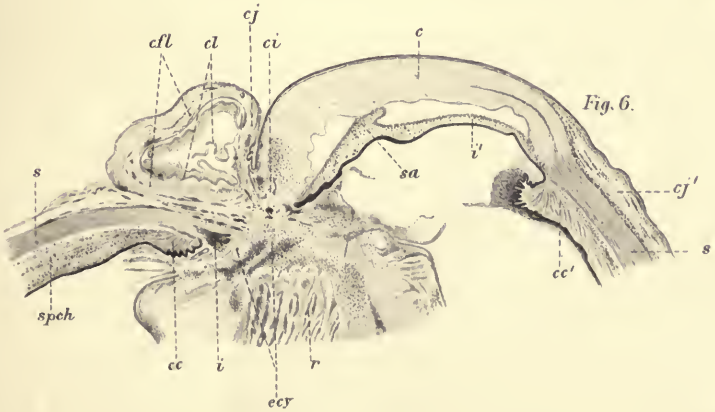

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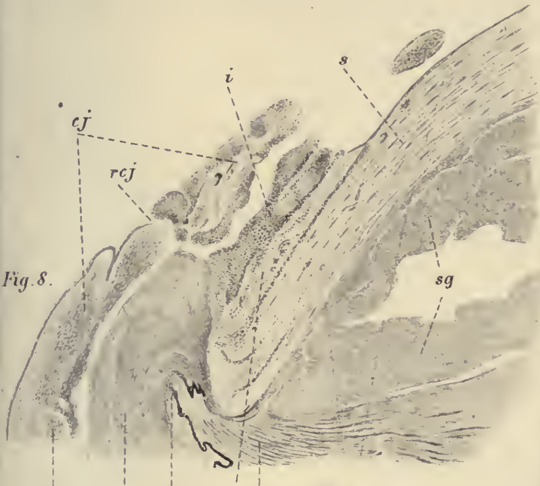

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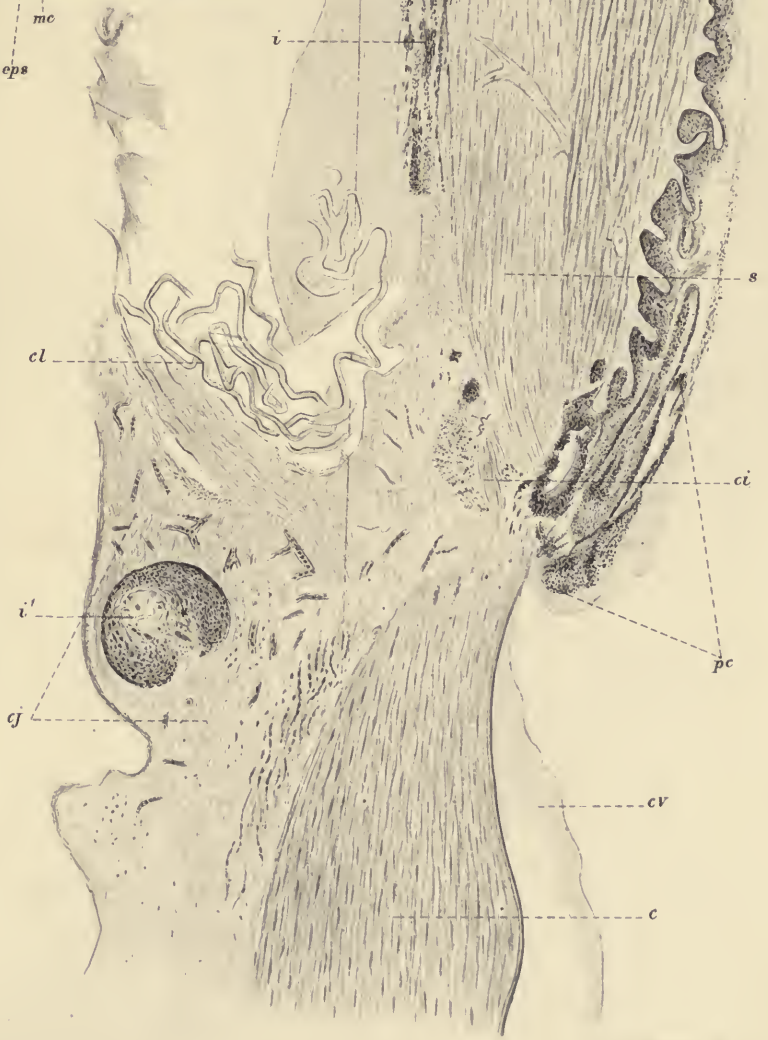

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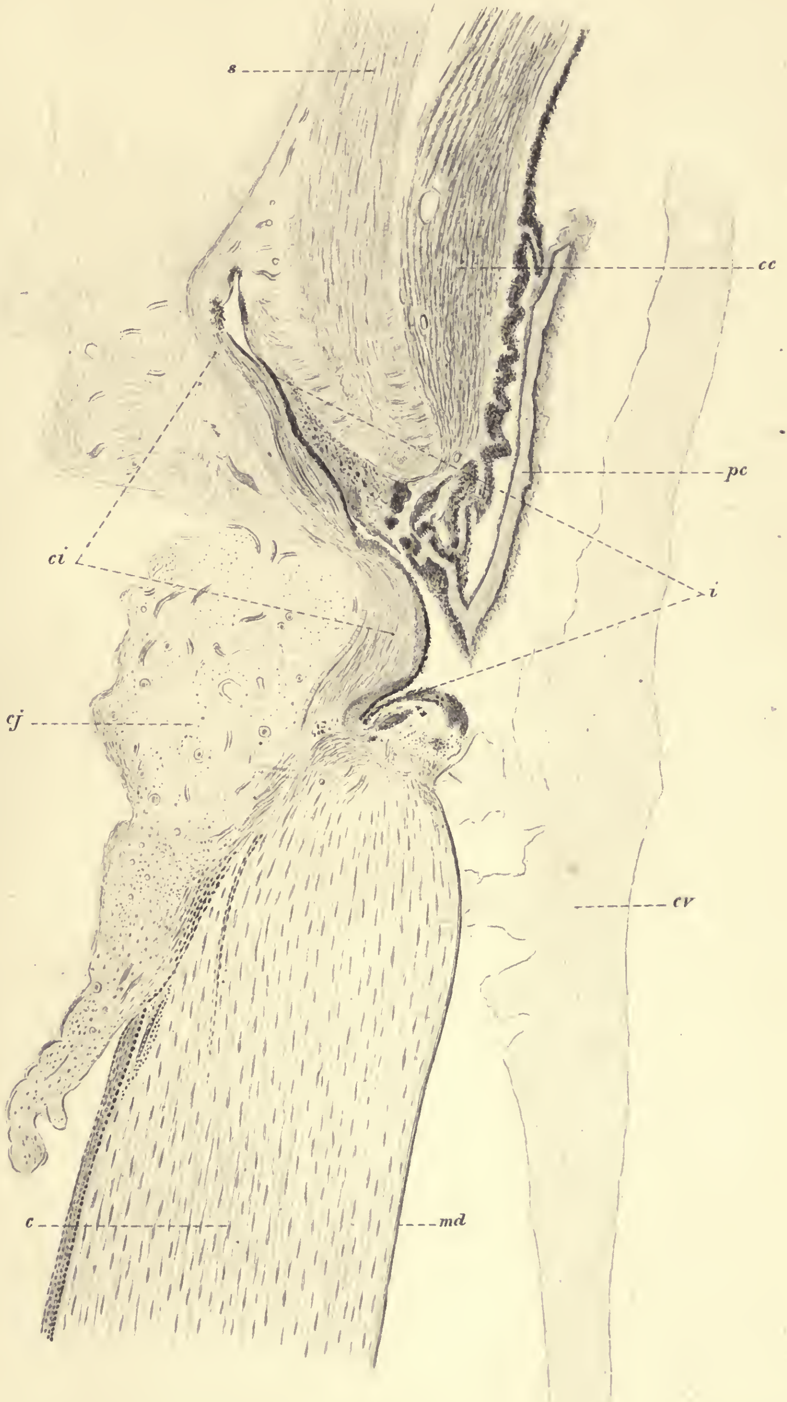

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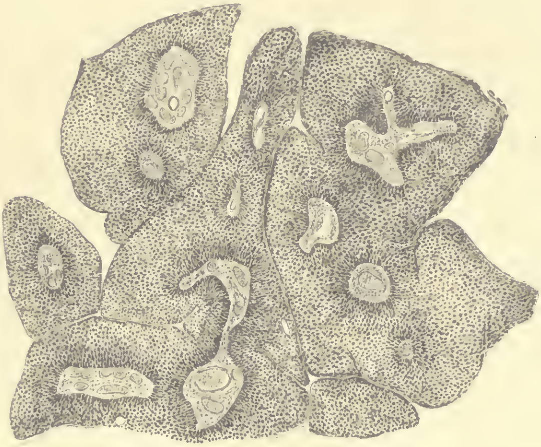

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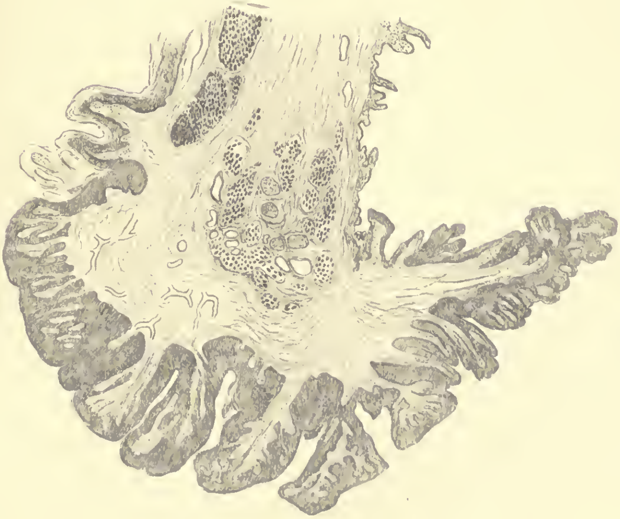

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